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HEAT AND SUMMER DIARRHEA *

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The theory that the extreme elevation of the temperature during July, August and September is the direct cause of summer diarrhea was very popular with the early American physicians. Booker[1] has given us an interesting history of the subject. Diarrhea in infancy as a summer disease was not recognized in Europe until recent times, but was accurately described by Benjamin Rush of Philadelphia in 1777. To this physician is also credited the theory that heat is the principal factor in the causation of the disease, but a careful reading of his article shows that he was inclined to view the disease as a modification of malaria. It was really Dr. E. Hornell of Philadelphia in 1823 who first clearly expressed the direct connection between heat and cholera infantum. He wrote:

> Whatever consideration attaches to irregularities in diet, inattention to cleanliness, difficulty of dentition, etc., I am disposed to consider the violent heats of the summer in conjunction with sudden aerial vicissitudes, or with exposure to a moist and vitiated atmosphere, as the most usual exciting cause of the complaint.

Another writer, J. P. Harrison of Louisville, Ky., in 1828, also considered atmospheric heat to be the general predisposing or remote cause of the disease and improper diet the most important exciting cause.

Dr. J. E. Cooke and others, at this period, denied the direct influence of atmospheric heat. Cooke declared that "there is evidently something more than heat necessary to the production of the disease."

Dr. Nathaniel Potter, 1833, also emphasized the importance of the high temperature and rejected marsh effluvia from the list of causative agencies. Eberle,[2] 1834, in discussing the various varieties and causes of diarrhea makes this statement: "There can be no doubt that high atmospheric temperature, by its direct influence on the system, is capable of giving rise to diarrheal affections." Eberle's teaching was dominant for fully fifty years in America, but additional factors were proposed by

* Read before the St. Louis Medical Society, May 31, 1913.

1. Booker: Arch. Pediat., 1901, xviii, 481.
2. Eberle: Diseases of Children, 1833, p. 215.

many writers. Meigs became the chief supporter of the theory of dentition and his authority had great weight with the general practitioner, and this teaching has not entirely died out even at present. Another prominent man who gave heat a prominent place in the etiology of summer complaint was Condie, who insisted, however, that the application of cold to the surface of the body or a sudden transition from a close and heated to a chilly and humid atmosphere will in many cases give rise to a diarrhea.

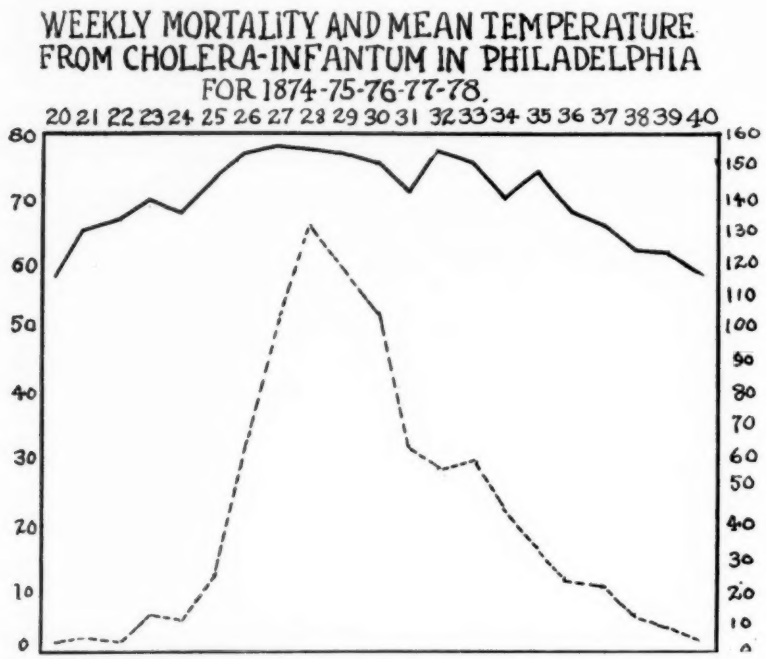

Fig. 1.—Drawn from Miller's statistics. No parallelism between the lower (mortality) curve and the upper (heat) curve.

The subject of heat and diarrhea received considerable discussion after the publication of an article by T. Clarke Miller,[3] who asserted:

> The condition under which cholera infantum appears, and the only condition essential to its development, is a continued high temperature day and night, and mean thermometer above 75 degrees with small daily range. This high and slightly varying temperature continuing from six to ten days will invariably, in our climate, bring cholera infantum, and the longer this condition of things continues the more numerous and intractable the cases become.

He collected the statistics of the mortality from diarrheal diseases from Philadelphia, New York, Boston, Baltimore, Cincinnati and Chicago, and gives the weekly mean temperature and the number of

3. Miller: Am. Jour. Obst., 1879, xii, 236.

deaths during the summer months. While there are numerous exceptions, the figures substantiate his theory to a remarkable degree (Figs. 1 and 2).

This position was vigorously combated by Woodbury,[4] who ventures "to differ *in toto* with him and other writers who adhere to the view that the degree of heat *per se* is sufficient to cause the disease." To quote further:

> We oppose it the more firmly because this teaching, which has now become fashionable, leads to erroneous therapeutic deductions; we are permitted to infer that a case resembles one of heat-stroke or thermic fever and, therefore, is to be treated by the extraction of bodily heat. Think of treating an adult with cholera morbus in this way. It is also leading us in the wrong direction for prophylaxis, for it teaches that if the baby can be kept cool it is safe.

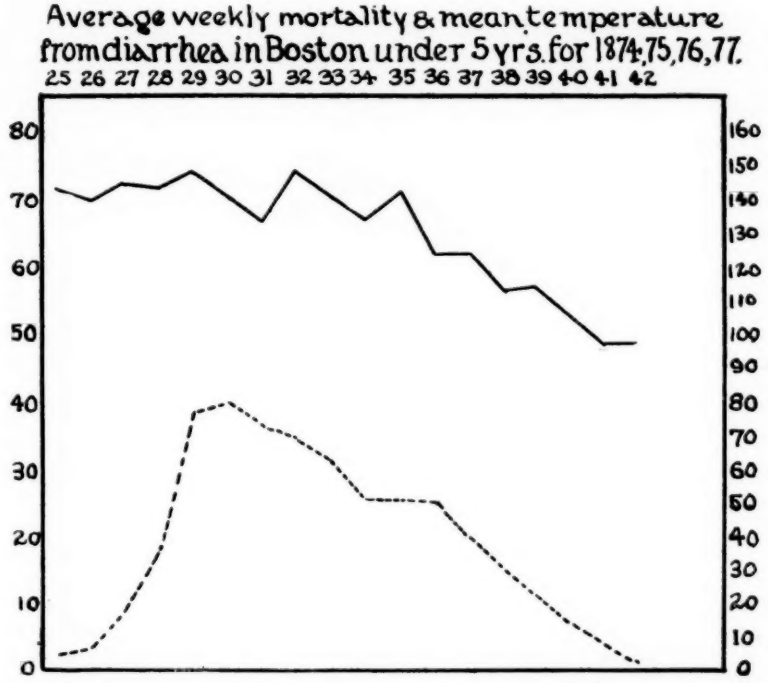

Fig. 2.—Drawn from Miller's statistics. Marked parallelism in the decline, but not the ascent of the curve.

He disparages the prophylactic measure issued by the New York Board of Health, "Wash your well children in cold water twice a day and oftener in the hot season." Furthermore, he insists that we should search deeper for the cause of summer diarrhea.

His principal objection to the heat theory may be summarized as follows: That children resist heat better than adults; that infants are often kept at a temperature of 75 degrees in the homes during the winter

4. Woodbury: Med. and Surg. Reporter, 1879, xli, 177.

for weeks; that the geographical distribution is different from what would be expected if heat was the chief cause, and that pathological considerations show that disease corresponds closely to cholera morbus in the adult.

In a reply to this article, Miller[5] emphasized his previous position, and points out some weaknesses in Dr. Woodbury's argument.

Atkinson,[6] in discussing the various causative agencies, filth, heat and improper food, is forced to the conclusion that there must be other etiological factors. Bigelow and others about this time were insisting

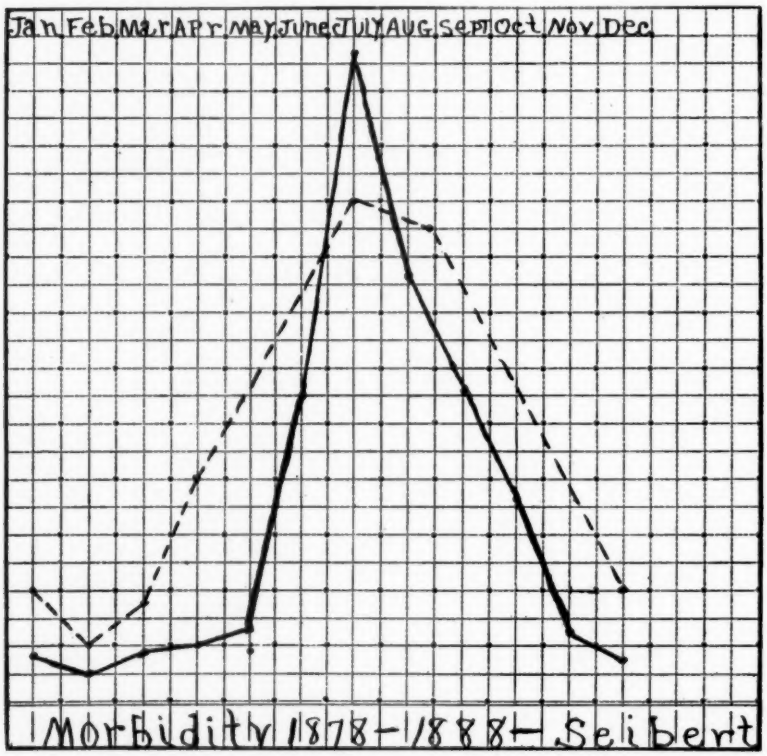

Fig. 3.—After Seibert. Dotted line, mean temperature; solid line, mortality. New York.

that cholera infantum must be clinically separated from enterocolitis and simple diarrhea. The older writers seem to have used these terms interchangeably, and much of the discussion has arisen from different writers studying the effects of heat on particular clinical forms. Here, however, we are concerned with all the diarrheal diseases of infants.

5. Miller: Med. and Surg. Reporter, 1879, xli, 282.
6. Atkinson: Med. and Surg. Reporter, 1880, xliii, 50.

An important study of the subject was made by Seibert[7] of New York. He gathered all the cases of acute gastro-intestinal catarrh in children under 5 years of age treated at the German Dispensary during a period of ten years, also the mortality of diarrheal diseases in New York for the same period and arranged the cases according to the different months. Curves of the number of cases and the average monthly temperature gave two figures which are well known (Figs 3 and 4).

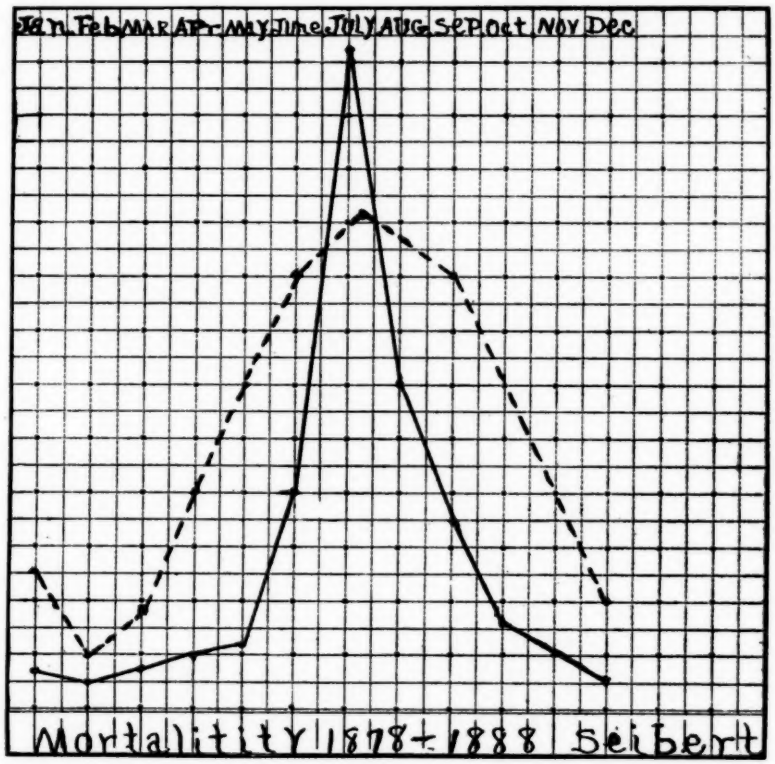

Fig. 4.—After Seibert. Dotted line, mean temperature; solid line, mortality. New York.

Seibert pointed out that the disease is present throughout the year, not frequently in January and February, but it does appear even in cold weather. The morbidity and temperature lines run in the same direction, but are not parallel. The greatest difference exists in morbidity between June and July, while the difference of temperature is not nearly so great as between May and June. He draws the remarkable conclusion from this study that the proportion of deaths is exactly the same in winter as

7. Seibert: Med. Rec., New York, 1888, xxxiii, 24.

in summer, during the hottest as well as during the coldest weather. He finds numerous discrepancies between the degree of heat and the morbidity and mortality. "One July may be hot and have many cases and the next may be exactly as warm and only have half the cases." The variability of the disease in different months and years does not correspond to the degree of heat. Several forcible examples are given, one of which may be quoted:

But the most striking example is July, 1884, during which time the temperature never reached 90 degrees, passed 85 degrees but on three days, and altogether reached 80 degrees but on eight days, showing a remarkably cool summer month, and yet we have 266 cases of summer-complaint; while the following August had sixteen days where the temperature reached 80 degrees, four

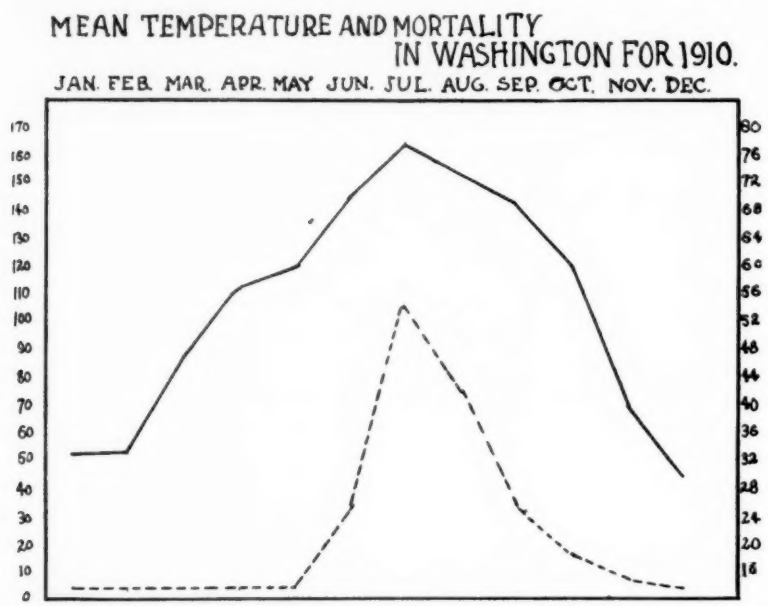

Fig. 5.—Solid line, mean temperature; broken line, mortality.

days with 85 degrees, and one with 90 degrees, and yet the number of cases is but 156, one-third less than in the preceding cooler July. I might keep on for hours and give you any quantity of evidence that very high temperatures do not favor the production of cholera infantum more than medium warm weather.

His conclusions are worthy of repetition:

1. Hot weather (either dry or moist) is not necessary for the epidemic appearance of summer complaint.

2. Warm weather (either dry or moist) showing minimum daily temperatures of not less than 60 F. brings on the epidemic appearance of cholera infantum invariably in every year, irrespective of the height of the maximal daily temperature, as in the latter part of June in every year.

3. Summer complaint loses its epidemic character as soon as the minimal daily temperature remains below 60 F., as in the latter part of October of nearly every year.

4. Therefore, this disease cannot be brought about by the direct working of high temperatures on the infantile body, but must have other causes.

The other causes, of course, are bacterial decomposition of milk and infections, the theory which had gained ground about this time. Dr. Miller[8] replied to this article without, however, offering any additional evidence.

American physicians generally adopted the conclusions of Seibert, and an earnest search for the other causes has resulted in the large and important work on the bacteriology of the summer diarrheas familiar to all. The work of Holt on the pathological anatomy and Booker on the

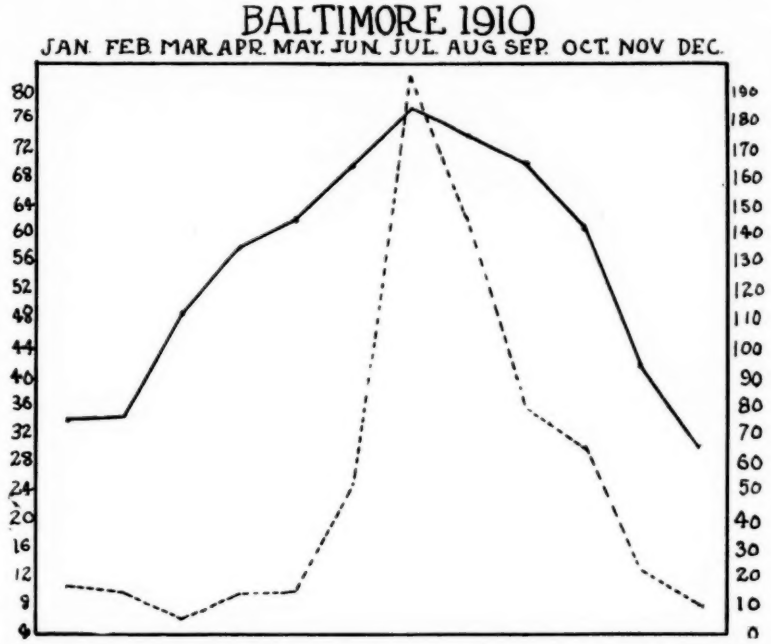

Fig. 6.—Deaths in Baltimore, 1910. Heat and mortality curves not parallel.

bacteriology of ileocolitis, strongly corroborated the earlier theories that virulent bacteria are the chief sources of the graver acute intestinal diseases. The position at that time was clearly stated by Holt:[9]

The chief exciting cause is something to the development of which two things have a fixed and constant relation, viz., a certain degree of atmospheric heat and the practice of artificial feeding. Both of these conditions are necessary. We believe the chief causative factor to be bacteria, and that these act in most cases by inducing changes in the food.

While we have learned in recent years that the ordinary saprophytic decomposition of milk is usually harmless, American physicians even

8. Miller: Med. Rec., New York, 1888, xxxiv, 59.
9. Holt: Keating's Cyclopedia Dis. Child., 1890, iii, 70.

to-day still cling to the position that certain kinds of bacteria and their products induce the most important pathologic changes in summer diarrhea. While the agency of heat is generally admitted in directly lowering the digestive and metabolic activity of the infantile organism, it is denied that the excessive heat alone without the bacterial or toxic factors would appreciably raise the ordinary morbidity of digestive and nutritive disorders in infants. We thought the question of heat had been answered, and while the bacteriology and toxicology were still unsettled we waited patiently for the difficult problem to be solved. The discovery of the different types of the dysentery bacillus and their wide distribution certainly promised to clarify the mooted question. We had already recognized from Booker's studies that a great variety of bacteria may be etiologically related to the disease. The study of the colon and dysentery

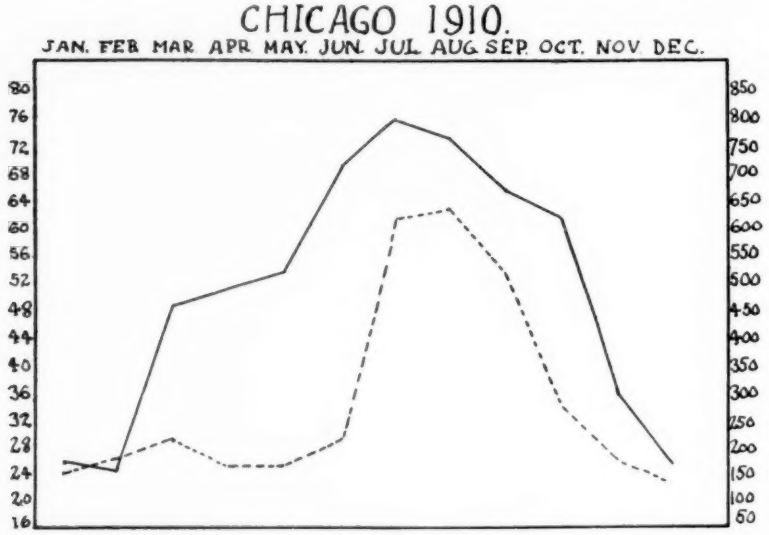

Fig. 7.—Deaths in Chicago, 1910. Note the absence of the sharp peak in the lower (mortality) curve.

group, the streptococcus group and the group classified under the liquefying bacteria, their pathogenic and toxicogenic powers, and the susceptibility and immunity of the infant offered an enormous field, and, as mentioned, we believed that we were approaching the solution of the intricate problem.

It seems, however, that the original controversy is open again, and what we had dismissed twenty years ago is brought forth in splendid attire and paraded as the long-sought causative agent. We are asked again to destroy the bogey of summer diarrhea by keeping the babies cool, and we expect our milk stations to close up and in their place conferences

will teach the mothers to give the baby several cool baths daily during the hot weather to abstract the retained atmospheric heat.

This view began with the teachings of Finkelstein, who all but discarded enteric infections from the nosology of infantile diseases. It was Rietschel,[10] however, who in a splendid monograph vigorously defends the old view that summer diarrhea depends almost entirely on the excessive heat of summer. In another article[11] he reiterates his former conclusions with increased vigor and offers additional evidence. His principal conclusions briefly stated are:

1. The summer mortality of infants is brought about by all the detrimental agencies which produce the general infant mortality, to which is added another injurious factor — the heat.

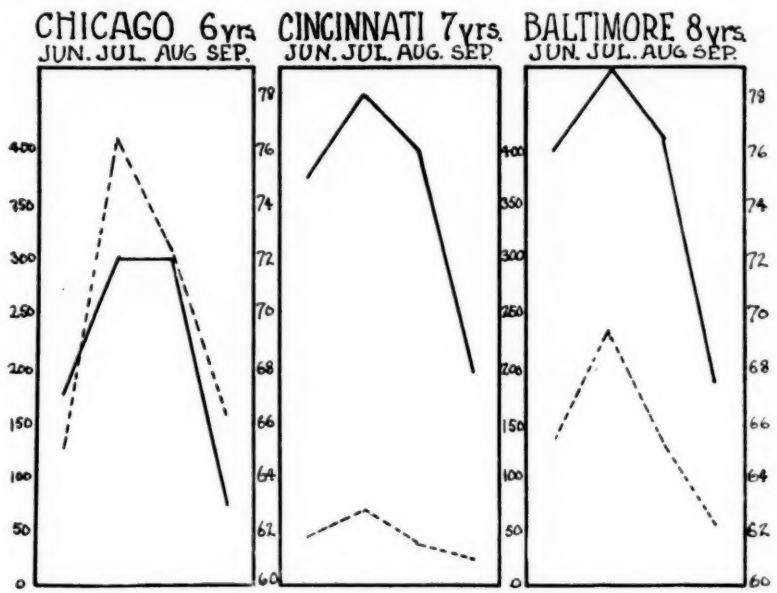

Fig. 8.—Drawn from Miller's statistics. This figure again shows that the heat and mortality curves do not correspond and vary in different cities.

2. The summer heat affects the infants through the high temperature of the living rooms, and the homes of the proletariat may be very hot even in a cool summer. The real danger of the heat to the infant lies in the rooms of the houses.

3. The action of the heat is a direct injury to the infant in producing hyperthermia, in the abstraction of water, overfeeding from the thirst, effect on the gastro-intestinal secretions and bacterial activity in the alimentary canal.

In the discussion of this subject by the *Gesellschaft für Kinderheilkunde,* 1911, several members protested against the dicta of Rietschel,

10. Rietschel: Ergebn. d. inn. med. u. kinderh., 1910, vi. Complete bibliography given, page 369.

11. Rietschel: Monatschr. f. Kinderh., 1910, p. 39.

but there can be no doubt that this old theory is favorably received in Germany.

What shall be our attitude on this question?

In order to assist in elucidating this subject the following study is herewith presented:

1. The mortality in different parts of the United States.

In order to ascertain what effect different climatic conditions have on the mortality of the disease the death-rate of diarrheal disease in infants under 2 years of age as given in the United States mortality statistics for the years 1907 to 1910, inclusive, were studied. For the purpose of comparison, several southern, several central and several northern cities were selected as given in Table 1.

TABLE 1.—MORTALITY FROM DIARRHEAL DISEASES IN DIFFERENT SECTIONS OF THE UNITED STATES

Cities		Deaths in				Death Rate per 100,000 in 1910
Southern	Population	1907	1908	1909	1910	
Mobile	43,642	43	59	42	48	93.0
Jacksonville	38,049	48	52	37	27	46.0
Atlanta	107,265	151	115	122	154	98.0
New Orleans	318,652	463	466	345	448	131.0
Wilmington	21,620	45	49	49	53	205.0
Charleston	56,402	119	141	111	142	241.0
San Antonio	64,275	172	216	200	291	298.0
Central						
St. Louis	661,666	482	443	476	596	86.0
Philadelphia	1,466,408	1,886	1,835	1,791	2,336	150.0
Louisville	229,599	110	73	65	64	28.5
Cincinnati	347,123	459	301	282	328	90.0
Indianapolis	227,698	190	158	130	177	75.0
Northern						
Boston	609,175	546	724	684	678	100.0
Detroit	367,494	395	374	369	581	123.0
Minneapolis	285,676	138	255	128	240	79.0
Milwaukee	322,513	347	410	467	441	117.0
Buffalo	386,724	598	586	533	563	132.0

It will be seen from Table 1 that there is no striking difference in the death-rate of diarrheal diseases in different parts of the United States. Latitude has little influence. The common practice of sending infants to the Northern resorts or to the sea shore finds little comfort from these figures. Louisville, Ky., seems to be the ideal summer resort for babies. This singular low death-rate in Louisville is inexplicable. In reply to a personal letter, Dr. Tuley attributed this low death-rate to good milk and effective educational work among the poorer classes.

A more striking difference is found in comparing the Eastern and Western cities.

Eastern Cities Death-Rate, 1910.		Western Cities Death-Rate, 1910.	
Portland	73.0	Seattle	31.8
Baltimore	114.0	San Francisco	40.0
Atlanta	98.0	Los Angeles	24.4

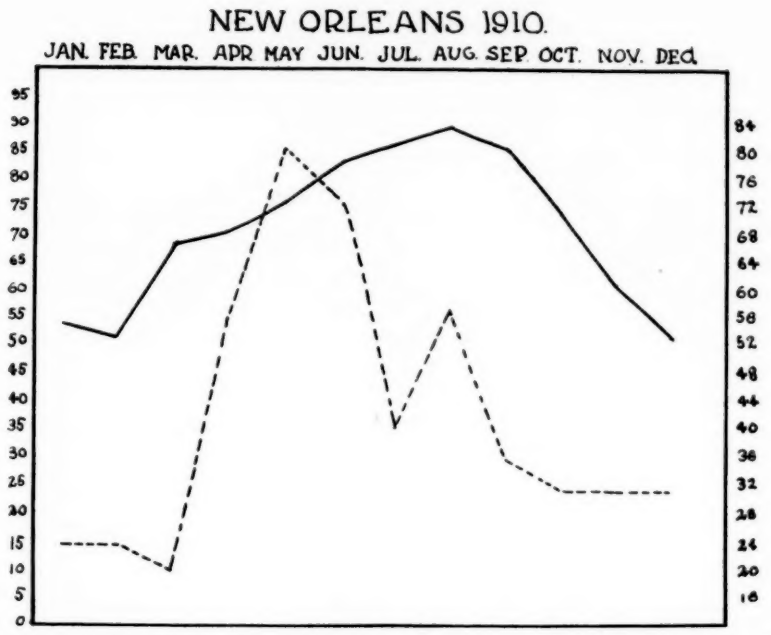

Fig. 9.—This shows the peculiar mortality curve (dotted line) shown by Southern cities. The peaks of the heat and mortality curves do not correspond, and the secondary rise in the mortality curve is characteristic.

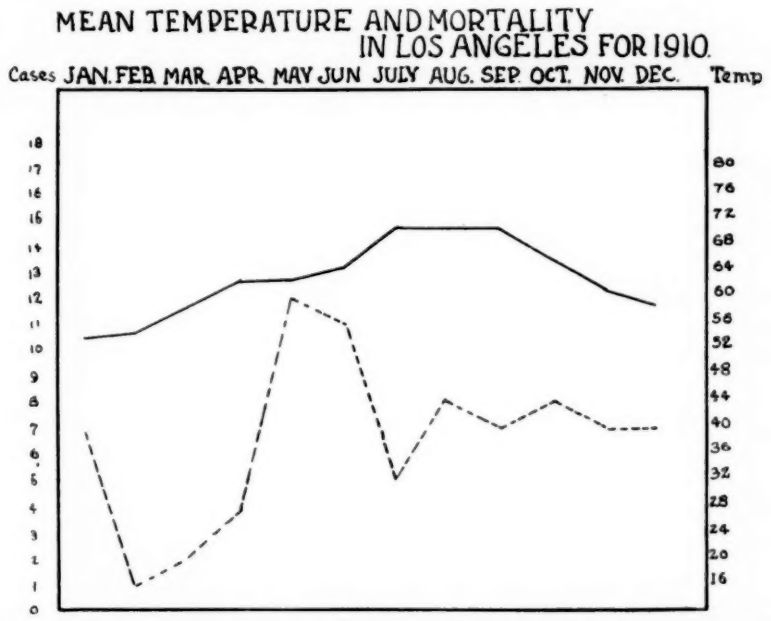
MEAN TEMPERATURE AND MORTALITY
IN LOS ANGELES FOR 1910.
Cases JAN. FEB. MAR. APR. MAY JUN JULY AUG. SEP. OCT. NOV. DEC. Temp
18 17 16 15 14 13 12 11 10 9 8 7 6 5 4 3 2 1 0
80 76 72 68 64 60 56 52 48 44 40 36 32 28 24 20 16

Fig. 10.—The mortality curve (dotted line) shows the peculiarity of Southern cities.

The equable temperature along the Pacific Coast certainly has a restraining influence in intestinal diseases.

It is interesting to arrange the large cities of the United States in the order of their ascending death-rates (1910).

Los Angeles	24.0	New York	123.0
Louisville	28.0	Detroit	123.0
San Francisco	40.0	New Orleans	131.0
St. Paul	70.0	Richmond	133.0
St. Louis	86.0	Philadelphia	150.0
Cincinnati	90.0	Chicago	159.0
Boston	100.0	Cleveland	169.0
Baltimore	114.0	Pittsburgh	177.0
Milwaukee	117.0		

It is obvious that there are other factors besides fluctuation of temperature that produce such wide variation in the deaths of infants. A comparison of the death-rate in 1910 in several states of the registration area shows no great difference north or south; the western states, however, show much lower figures.

California	39.0	New Jersey	117.0
Colorado	65.0	New York	106.0
Connecticut	103.0	North Carolina	137.0
Indiana	78.0	Ohio	84.0
Maine	87.0	Pennsylvania	144.0
Maryland	103.0	Rhode Island	153.0
Massachusetts	121.0	Utah	49.0
Michigan	83.0	Vermont	57.0
Minnesota	64.0	Washington	37.0
Montana	59.0	Wisconsin	72.0
New Hampshire	93.0		

Basing the death-rate of infants on the whole number of inhabitants does not give very good figures for comparison, but it is the best we can do at present.

The mortality is especially high in manufacturing cities. The following are examples:

Fall River, Mass.	373.0	East Chicago	422.0
New Bedford, Mass.	324.0	Gary, Ind.	404.0

The mortality in rural districts is generally less than in the large cities. Thus we find that in 1910 the death-rate from diarrheal disease in the registration area was 100.8; the cities gave the mortality as 117.8; the states, 98.7; while the rural part of the states was 77.3. In 1911 in the state of Missouri, the death-rate in St. Louis was 85 per 100,000; five adjoining counties taken at random in the central part of the state had a death-rate of 51 per 100,000 inhabitants. On the other hand, three counties in the southeastern part of the state had a death-rate of 130.

The death-rate in Missouri was 63.62 in 1911. This compares favorably with most states. The death-rate in Michigan in 1910 was 83, and

in Minnesota 64 during the same year. The rural part of Missouri is a good place to keep infants during the summer.

The relationship of the mean monthly temperature and the number of deaths in a number of cities of the United States is shown by the

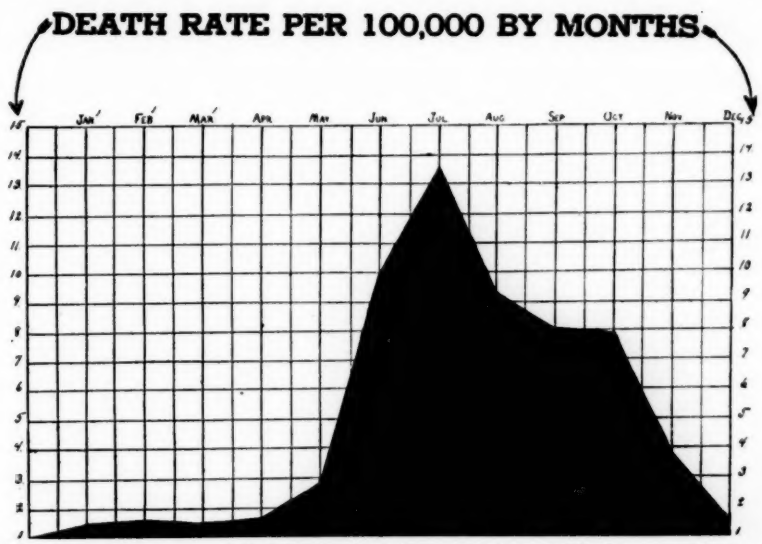

Fig. 11.—The curve of the deaths in Missouri, 1911. Note the secondary elevation in October. The last drop corresponds to the appearance of frost.

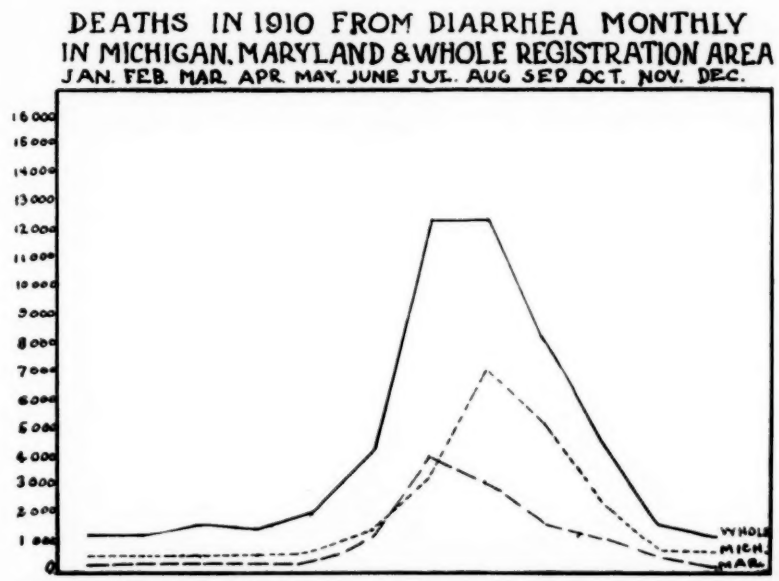
DEATHS IN 1910 FROM DIARRHEA MONTHLY
IN MICHIGAN, MARYLAND & WHOLE REGISTRATION AREA
JAN. FEB. MAR. APR. MAY. JUNE JUL. AUG SEP OCT. NOV. DEC.
WHOLE
MICH.
MAR.

Fig. 12.—The curve of deaths over the whole registration area shows a plateau and not a peak. Note the difference in the time in the highest mortality in a Northern and Southern state.

curves (Figs. 5, 6 and 7). The curves are drawn from the temperature and death-rate in 1910. The curve of Chicago shows a peak flattened at the top. Three cities are compared in Figure 8, taken from Miller's figures.

In all northern and eastern cities there is a striking similarity. Southern cities show a different curve. The apex of the heat curve and mortality curve do not correspond, and a secondary elevation is peculiar (Figs. 9 and 10). The peak of the curve is not so sharp as that of the northern cities. A similar curve is exemplified by Missouri, 1911 (Fig. 11). A high death-rate continued until the last of October, although the temperature had fallen very much. A similar condition occurred in

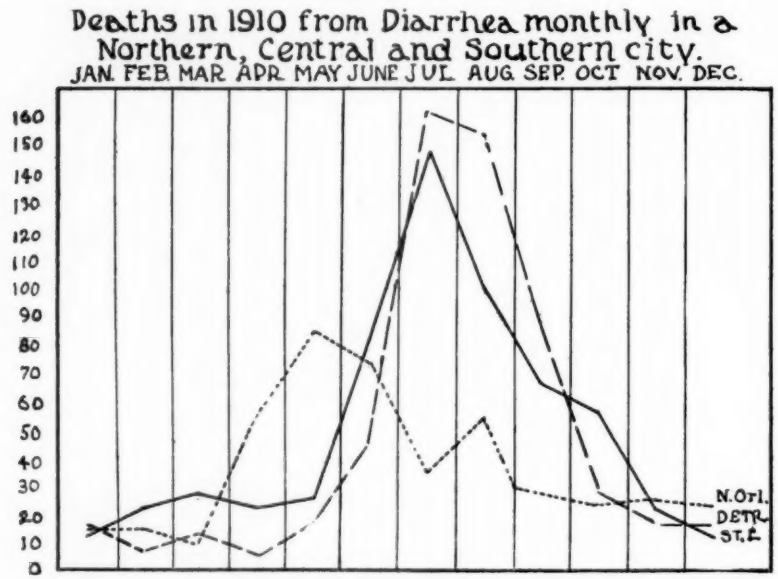

Fig. 13.—This shows the different configuration of the curves of mortality of different cities.

1912. Two states are compared with the registration area in Figure 12; three cities are drawn in Figure 13. The deaths in San Francisco show a singular curve. The mean temperature in October rose slightly above 60 F., yet there is a sharp peak (Fig. 14).

The intimate relationship of a high temperature and the morbidity and mortality is clearly shown by the classical curves of Seibert (Figs. 3 and 4).

However, his statement that the death-rate of diarrheal diseases is practically the same in winter and summer, while apparently substantiated by the configuration of the curves, is really disproved when his figures are properly analyzed. Assuming that the number of cases

treated at the German Dispensary approximately represents the morbidity of the disease, we find that the number of deaths is relatively much larger in the summer. The total number of cases seen during the four winter months was 620; the total deaths during the same period in New York were 1,476—that is, the ratio of the morbidity at the German Dispensary to the total mortality was 1 to 2.4. In July the ratio was 2,443 to 12,468, or 1 to 5. The death-rate, therefore, is at least twice as high during the summer months as in the winter months, a conclusion which corresponds to the common impression of practitioners. This difference is shown in the curves in Figure 15 in which Seibert's mortality figures are divided by two.

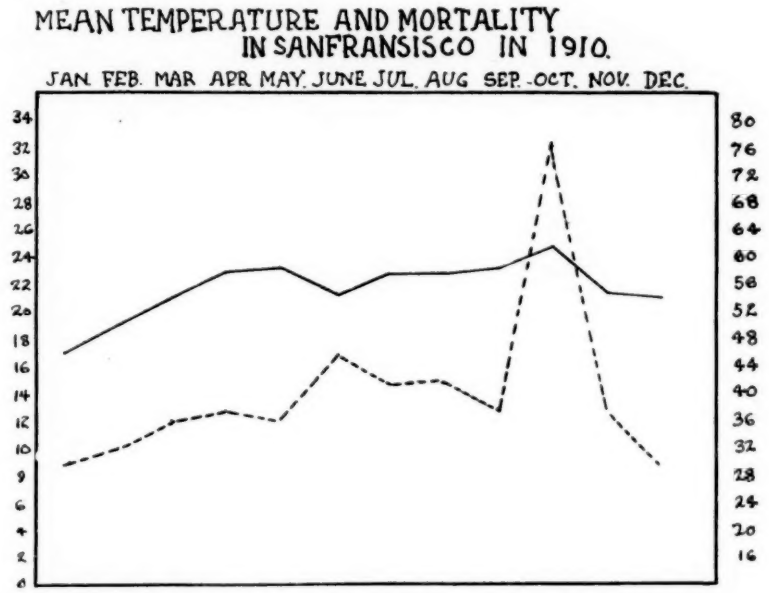

Fig. 14.—San Francisco has a singular curve of mortality; its peak (dotted line) corresponds with the peak of the mean temperature (solid line).

The relation of heat and mortality is also shown clearly by the figures published by Miller. Figures 1 and 2 are based on these, and plainly show why he was so firmly attached to the principle that heat is the essential factor. The peculiar phenomenon of a rise in mortality during the hot months is not peculiar to the United States. The rule also holds good in Europe, as has been repeatedly shown. For illustration, a chart of Budin of Paris is appended (Fig. 16).

All these studies relate to the average monthly temperature and mortality. In order to study the relationship more closely the daily number of deaths and the temperature have been compared. Copies of the curve of Finkelstein (Fig. 17) and Willan (Fig. 18) are instructive

in that they demonstrate that more deaths occur on the hot days: The curve published by the New York Milk Committee (Infant Mortality and Milk Stations, 1912), a part of which is herewith given (Fig. 19), does not agree so fully.

I have made similar curves of the daily mean temperature and deaths in St. Louis for two summers (Figs. 20, 21, 22, 23, 24 and 25).

The striking exception in August, 1911, is worth special mention. September, 1912, had some peculiarities not accounted for by the heat. Figure 26 shows deaths in St. Louis and in the state compared.

It is obvious that while many hot days show a high death-rate there are other equally as warm days in which no corresponding increase in

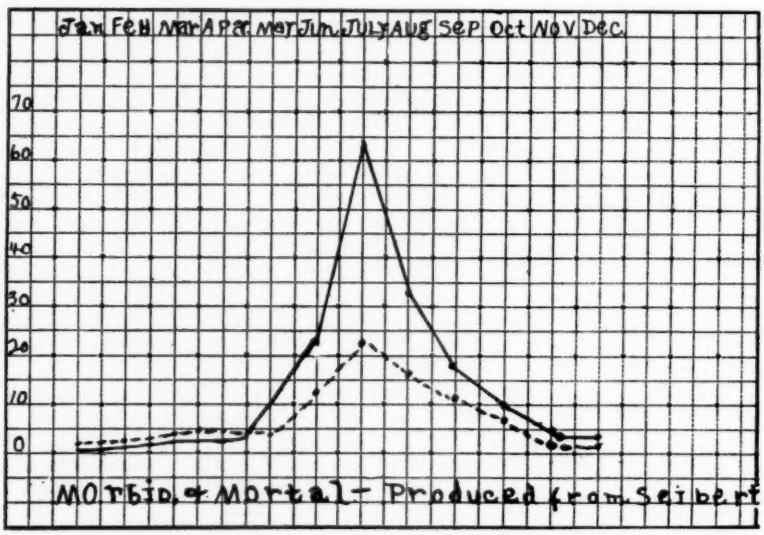

Fig. 15.—The lower curve represents the relative morbidity; the upper, the mortality. Drawn from Seibert's numbers. It will be seen that the mortality curve is much higher during the summer; that is, the death-rate is higher.

the deaths is found. Warm periods and many deaths agree closely in the beginning of the summer, but this is not true later in the season.

For the purpose of learning how long the infants who died during the days of high mortality had been sick, the mortuary records of St. Louis were studied. The death certificates give the length of the illness and from these a conclusion can be drawn whether short illnesses, such as convulsions, heat stroke, etc., make up the greater part of the death-rate.

Table 2 gives the age of the baby in months (first number) and the length of the illness in days (special number). The length of each line roughly gives a curve of the deaths. The mean temperature is affixed. The illnesses directly due to heat stroke are very few. Two cases probably

occurred on July 5. Rapid deaths from heat-stroke even in St. Louis are too few to modify the mortality curve appreciably.

In Figure 27 three months are studied to determine the onset of illness. It is perfectly clear that both morbidity and mortality in June and July are distinctly influenced by the height of the temperature. In August and September this relationship is not nearly so clear. Cool periods may be attended by a rise in mortality (Fig. 21).

TABLE 2.—DEATHS IN ST. LOUIS IN JULY, 1911.

Day	Mean Temp.	Age and Duration of Illness
1	86	6-6,* 18-2, 4-7, 9-3, 9-8, 1-10
2	88	16-37, 9-1, 1-4, 11-1, 16-3, 8-1, 9-5, ½-2
3	89	4-3, 3-8, 2-10, 1-4, 8-2, 1-7, 14-3
4	91	4-14, 4-18, 2-3, 21-8, 1/6-5
5	89	2-3, 2-1, 2-8, 6-1, 9-14, 8-7, 2-39, 4-12, 21-14
6	85	2-4, 4-3, 6-12, 6-2, 15-2, 9-10
7	85	27-5, 14-3
8	83	5-2, 14-2, 10-1, 5-10, 7-3, 4-2, 9-5, 4-4
9	83	13-19, 6-1, 14-2, 4-4, 12-4, 4-3, 7-30, 9-60, 20-6
10	84	4-20, 23-4, 3-21
11	86	5-5, 12-4
12	81	17-4, 6-60, 10-1
13	79	12-43, ⅔-10, 2-35
14	80	5-5, 20-5, 15-6, 4-2, ⅔-20, 2-10, 2-60, 1-1
15	82	6-14, 6-2, 7-6, 2-14, 12-2, 11-9, 12-18
16	75	9-13, 20-3
17	73	4-7, 3-4
18	72	4-13
19	73	6-10, 12-24, 8-5, 8-30, 4-34
20	76	1-9, 3-1, 8-12
21	81	9-4, 11-3, 2-4
22	79	7-7, 4-27, 10-15
23	78	16-22
24	69	7-6, 8-18
25	68	9-2, 11-10, 6-25, 5-30
26	69	10-14, 10-26, 6-11, 19-5, 7-1
27	76	18-4, 4-60, 4-20, 17-2
28	74	28-10, 9-5, 3-60, 1-2
29	75	13-9, 5-9, 10-30, 10-14, 9-30
30	76	24-4, 8-9
31	78	10-3, 5-2, 3-18, 3-20, 6-2, 2-10

* First number, age in months; second number, length of illness in days.

Clinical experience shows that the cool weather in September and October may be accompanied by an increase in diarrhea (Fig. 25).

America is known as the country of extreme variations in temperature, and to this physical character has been ascribed the great prevalence and high mortality of diarrheal diseases. As various parts of the United States exhibit this variation in temperature in a different degree, an inquiry into the death-rate and heat variation may be instructive. Figure 28 is based on the difference in temperature of the winter months (December, January and February) and the summer months (June, July and August) in 1910 in several cities of the United States.

Observation in St. Louis has convinced me that it takes two or three days of very warm weather to heat the interior of our brick houses to the extreme temperature. When this heating is accomplished the daily temperature variation is much less in the houses, and sleep, even for adults, is difficult at night. These are trying times for infants suffering from vomiting and diarrhea. Liefman and Lindman have attempted to prove

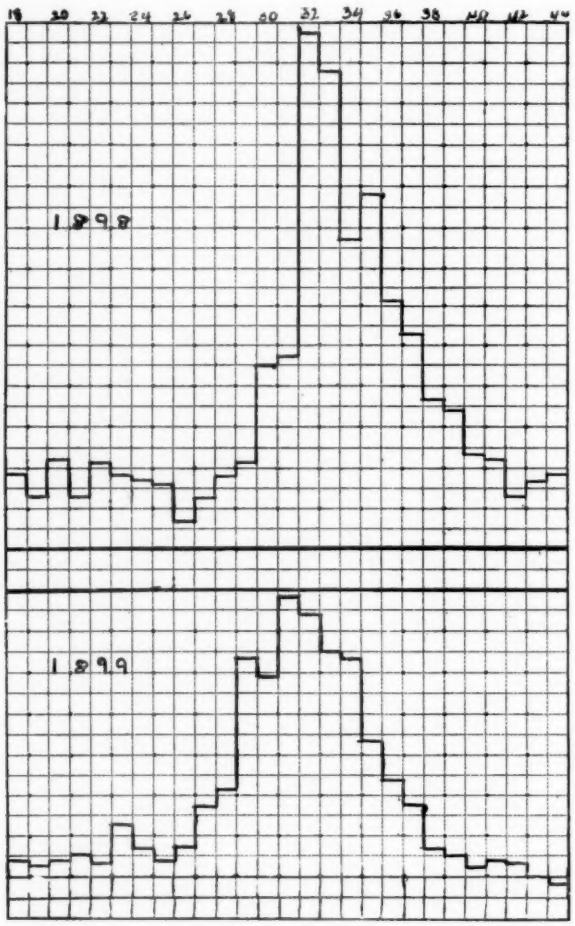

Fig. 16.—Summer diarrhea in Paris. After Budin.

that the mortality is higher after the second day of hot weather, but a special effect on the second and third day cannot be discovered in the curves here presented.

From this study, I must conclude that the direct effect of excessive heat is inimical to infants sick with gastro-enteric diseases, and the mortality is higher in the summer. This is really the common clinical

impression, that diarrhea is more protracted and more fatal in the summer than in winter.

An entirely different question is offered by the prevalence of diarrheal diseases in the summer. Does the excessive heat produce diarrhea by its direct effect on the organism? Miller believed that it does, and Rietschel and others have attempted to prove it. My own study is unequivocally against it.

In the first place, numerous observations on premature infants kept in incubators which have been kept too warm (Budin,[12] Zahorsky[13]) have failed to note diarrheal tendencies. The symptoms are flushing of the

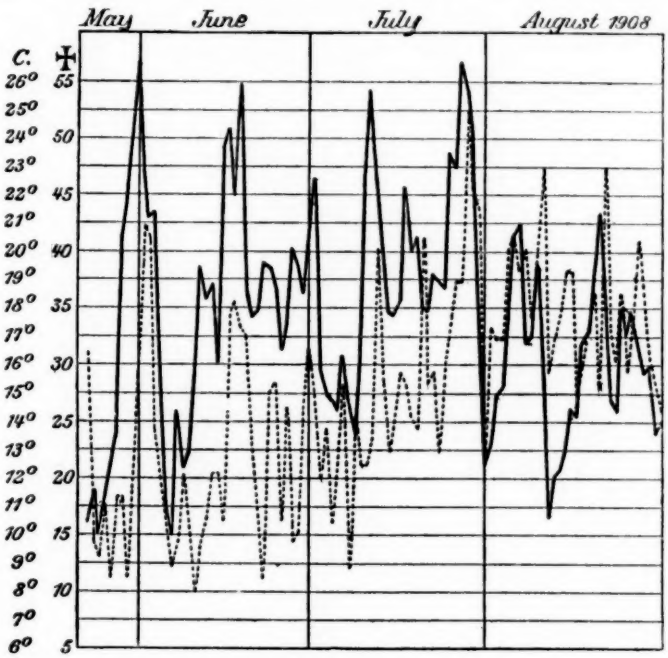

Fig. 17.—The curve of Finkelstein, showing daily deaths and mean temperature in Berlin, 1908.

skin, perspiration, anuria, rapid shallow respiration and an elevation of temperature.

We are familiar with the direct effects of heat on infants in St. Louis. On numerous occasions the temperature in the houses in the last fifteen years has reached 92 to 95 F. The babies lie quiet and the symptoms are the same as those noted in premature infants. A few times conditions have been such as to produce marked thermic fever — even heat stroke — among infants under my observation. A memorable instance occurred in

12. Budin: Le Nourrisson, 1900, p. 18.
13. Zahorsky: St. Louis Courier Med., 1905, xxxii, 68.

1897 in the Bethesda Foundling Home. This incidence was referred to briefly in a general discussion on thermic fever in infants (Zahorsky[14]). The records were accidently destroyed soon afterward and the cases were never properly placed in the literature. To quote from the article mentioned (page 144):

This summer I had the sad opportunity to witness four fatal cases among the infants at the Bethesda Foundling Home. Altogether about forty cases of marked thermic fever were noted in this institution this summer; and I am convinced that in former years such cases also occurred, but were not diagnosticated as such. On the night of June 11, 1897, an infant had an eclamptic seizure. A few minutes afterward another infant was attacked in the same way. The rectal temperature was 110.6 F. in the one, and 111.4 F. in the other. They were imme-

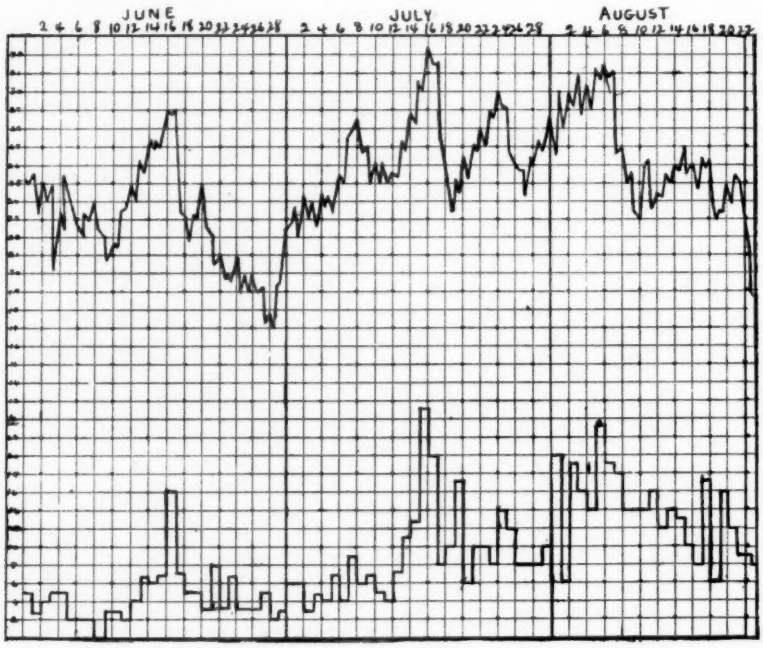

Fig. 18.—After Willim. Mortality from gastro-enteric diseases in Breslau, 1904.

diately placed in a cold bath by the nurses. When I arrived one was dead and the other died in a few minutes, although cold baths and stimulants were freely used. I found at the same time six other infants who had fever ranging from 102 to 107 F.; all these, however, were saved. On careful inquiry and examination no evidence of acute gastro-intestinal infection could be elicited, although vomiting had been a symptom in a few cases.

The post-mortem and blood examination showed clearly that thermic fever only could explain the disease. On the evening of July 8, again several cases occurred and one more death with hyperpyrexia. About one-half of the infants in the nursery during this period of heat showed febrile movement ranging from 101 to 108 F., which could not be attributed to any form of infection. On the

14. Zahorsky: Pediatrics, 1898, v, 144.

morning of July 11 the atmospheric temperature fell 15 degrees, and with it all the fever which had persisted for four days in spite of care, disappeared as if by magic. About fifteen cases more, only two hyperpyrexial, occurred during the rest of the summer.

From page 150 another quotation:

The first series of heat strokes that were noticed last summer occurred during the sudden rise of terrestrial temperature June 8 to 12, when in four days the

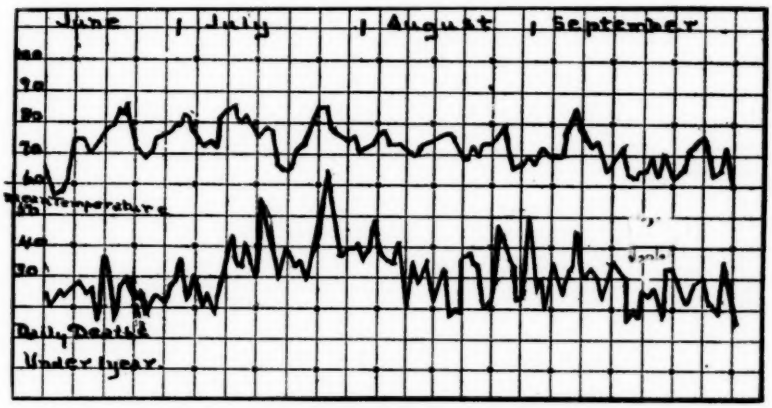

Fig. 19.—Mortality and mean temperature in New York City, 1912.

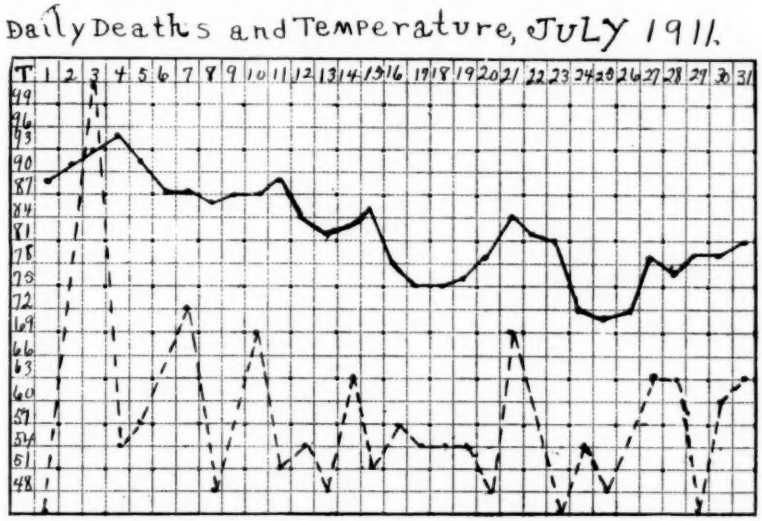

Fig. 20.—Daily mean temperature and deaths from diarrheal diseases in St. Louis, July, 1911.

daily maximum increased twenty-five degrees. It was during this period that two fatal cases occurred at the Bethesda Foundling Home. On June 17 the atmospheric temperature reached 96 degrees and several cases occurred again with one death. One case in private practice was studied at this time. But in this instance the additional heat of a baker's large oven in the basement added

to the temperature. The next series, and most general, occurred July 8 to 11, when in four days the atmospheric heat rose from 89 to 97 F. July 10 was memorable, as thirty babies at this home had temperatures ranging from 100 to 107 F., in spite of the free administration of water and the best of care. The nurses and physicians were constantly busy giving the overheated little ones a bath. July 11 the temperature fell 20 degrees and a remarkable result occurred.

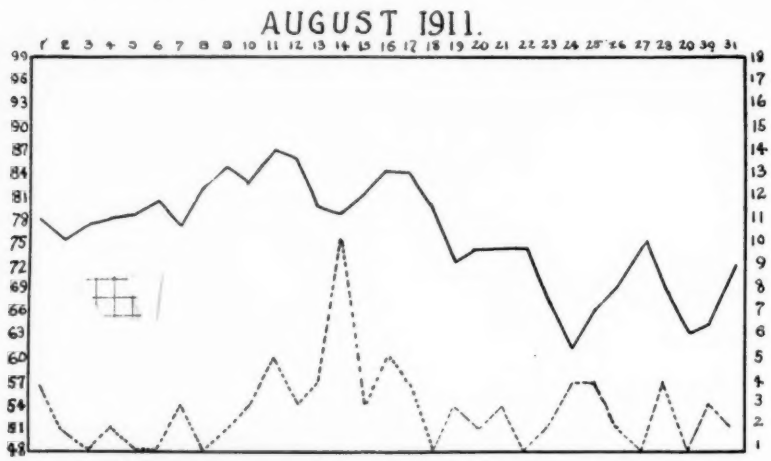

Fig. 21.—Daily mean temperature and deaths in St. Louis, August, 1911.

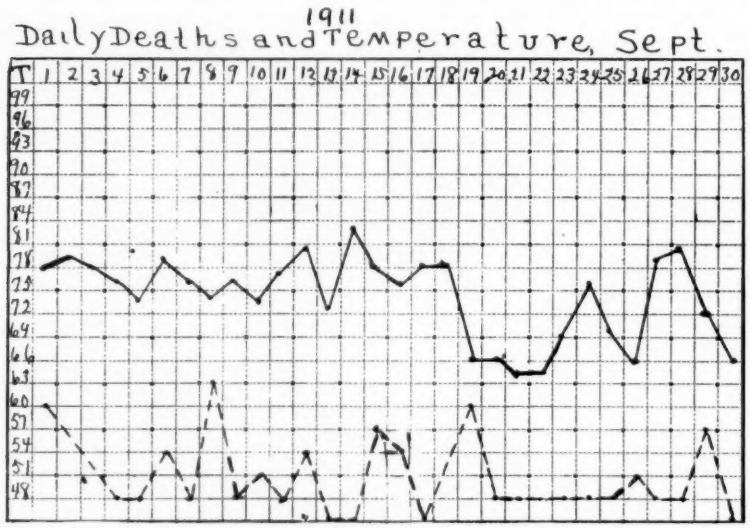

Fig. 22.—Daily mean temperature and deaths in St. Louis, September, 1911.

The thirty infants who had been having febrile movement for three days, some distinctly hyperpyrexial, became free from fever inside of twenty-four hours. This was so striking, and accompanied the fall in general heat so closely, that it forms a powerful argument to sustain the thermic rather than the fermentative causation of the pyrexia. The general average for this period was 88 degrees.

The next cases occurred during the latter part of July and the first week in August. The average mean temperature for this period was 88 degrees again, with a maximum of 97 degrees. Two cases with a temperature of 107 degrees developed. The high temperatures of August and September were not accompanied by thermic fever, although it occasioned, no doubt, more pyrexia among the many cases of gastro-intestinal infection which were treated at that time.

A few years later a similar epidemic occurred in the same institution, but was much less violent. Many times since the rectal temperature of infants on hot evenings has been 1 to 3 degrees above normal, and yet in all these observations no diarrheal tendencies have been observed. Thermic fever and diarrhea are distinct diseases and have little relationship.

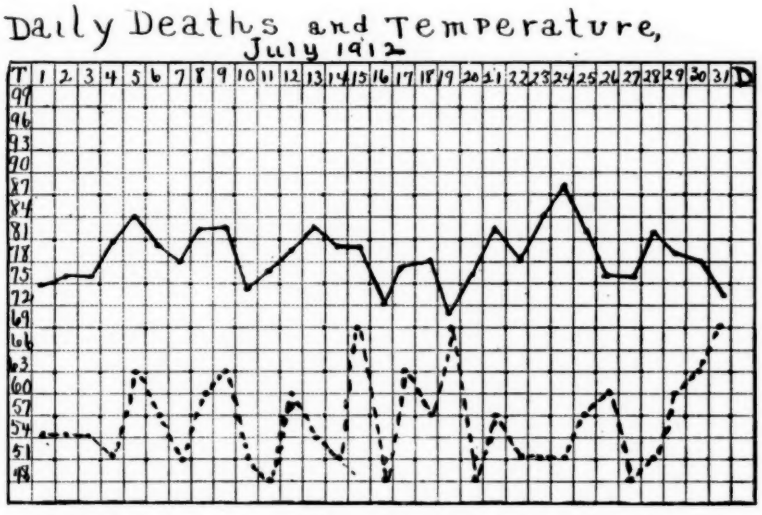

Fig. 23.—Daily mean temperature and deaths in St. Louis, July, 1912.

Experimental evidence against the heat hypothesis is found in the work of Genersich,[15] who ascertained that a room temperature of 28 to 30 C. produced an elevation of the rectal temperature in infants, but no gastro-enteric disturbance was observed. A more exhaustive study by Kleinschmidt[16] had a similar negative result and his studies in metabolism revealed nothing abnormal. He is positive in denying the inimical effect of heat on the digestive apparatus of infants.

Helmholz[17] studied the effect of the temperature of dwellings in Chicago, and the occurrence of diarrhea and published the following

15. Genersich: Monatschr. f. Kinderh., 1910, 183.
16. Kleinschmidt: Monatschr. f. Kinderh., 1910, p. 455.
17. Helmholz: Tr. Am. Assn. for Study and Prevention Infant Mortality, 1912.

instructive table. The outbreaks of diarrhea occurred in days with the maximum temperature indicated.

Temperature F.	70-74	75-79	80-84	85-89	90-100
Cases	7	9	9	5	-2
Percentage	22	28	28	10	6

He concluded that the heat *per se* was not a great factor in causing diarrhea or fever.

Knox[18] studied the effect of heat in the dwellings in Baltimore without observing any ill effect on the well being of infants.

Alter all, the subject needs a greater study of clinical cases. Acute cases of diarrhea should be studied more carefully as to the mode of

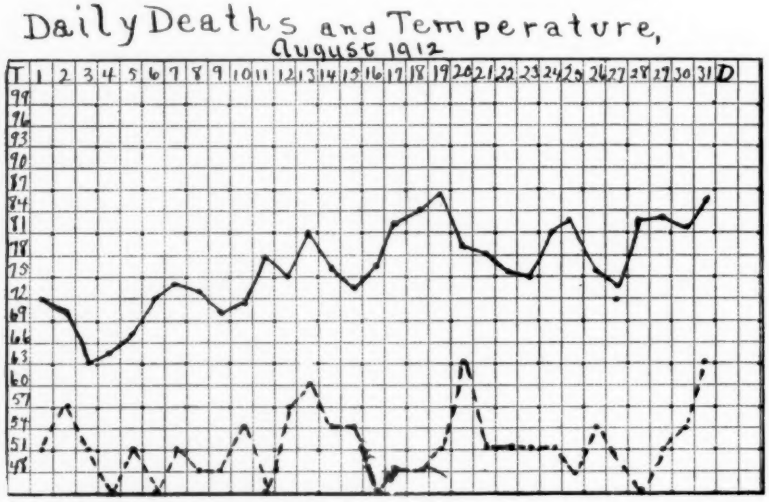

Fig. 24.—Daily mean temperature and deaths in St. Louis, August, 1912.

onset and the attendant atmospheric conditions. In the following are tabulated all the cases seen by me in private practice during the summers of 1911-12. The date of onset is compared with the atmospheric temperature.

CASES SEEN IN PRIVATE PRACTICE

Mean temperature ..	61-65	66-70	71-75	76-80	81-85	86-90
Cases	5	12	12	38	29	13
The ratio	1.0	1.4	1.4	7.6	5.8	2.6

If we find the ratio of the number of days with the above temperatures to the number of days in the whole period which is as follows:

1 1.3 2.7 4.3 3 1

the conclusion is inevitable that more cases of diarrhea occur during the warm days, but the greatest number occur when the mean temperature is

18. Knox: Tr. Am. Assn. for Study and Prevention Infant Mortality, 1912.

between 75 and 80 F. The number of cases, however, is too small to draw any positive conclusions.

My experience with cases of thermic fever reveal the fact that it is the older infants — 6 months to 15 months — who suffer the most severely from the heat. Thus in the epidemic mentioned, younger infants did not react at all or much less. This was attributed at the time to the fact that the older infants received undiluted cow's milk, a food having a high nutritive ratio, while the younger infants received diluted milk. That is, in hot weather a food very rich in protein predisposes to hyperthermia. This is only the rational deduction from physiologic principles, since the body has no storehouse for protein. However, at the time when our older infants were reacting with the remarkable cases of hyperthermia, no diarrheal tendencies were observed among the younger infants.

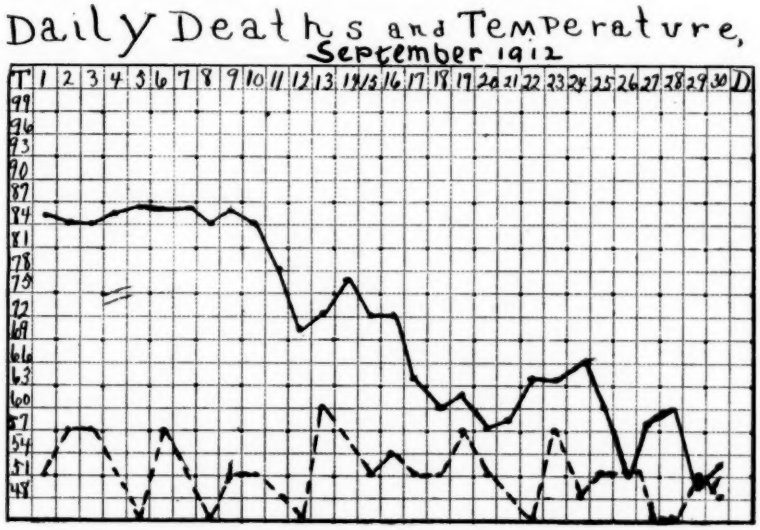

Fig. 25.—Daily mean temperature and deaths in St. Louis, September, 1912.

A study of the mortality from the Bethesda Foundling Home is appended. The mortality for the whole time is shown in Figure 29. The mortality after pasteurized milk and effective refrigeration methods were adopted was much less. We have had some diarrhea since adopting these measures, but none of the severe epidemics which almost wiped out all our babies in previous years. We have had some very hot weather in the last five years, and cases of hyperthermia have occurred, but all severe forms of diarrhea were absent.

A single example of diarrhea apparently produced by excessive heat alone is exhibited by Rietschel, but the clinical and experimental evidence is overwhelmingly against him.

The form of the disease which is mostly to be feared is the dysenteric type, commonly grouped under the name of ileocolitis, or infective diarrhea. Here the protracted febrile cause, in spite of favorable changes in temperature, the character of the stools, the high mortality and post-mortem findings plainly demonstrate its infectious origin. What per cent. of the whole number of cases are caused by this infectious form is not known, but judging from my own experience the majority of deaths in infants over 6 months of age are traceable to these infections. The typical cholera infantum is rare.

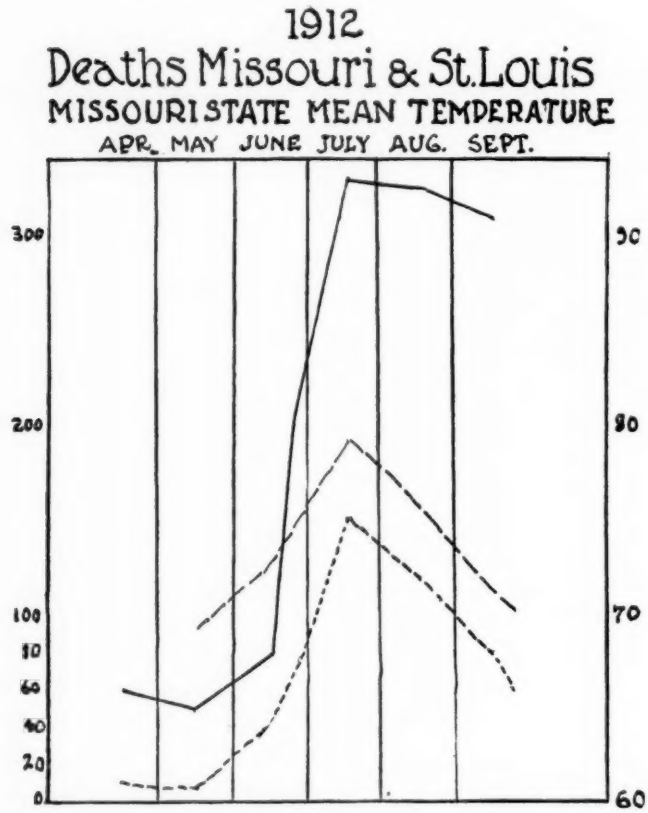

Fig. 26.—Curves comparing the deaths in Missouri State and St. Louis City.

The only forms of summer diarrhea which might be induced by excessive heat are the fermental diarrheas and the intoxications. Even in these cases the evidence that heat alone is more than a predisposing cause is altogether insufficient.

In the early period of July the correspondence between the height of the temperature and the morbidity is very striking. This parallelism may be observed in Figure 20. It is less marked in August or September.

In general, the high basal line of mortality corresponds to the increase in diarrheal diseases in the summer as shown in the curve drawn from Seibert's figures. The sharp elevations correspond to the increased mortality produced by the heat. Liefman and Lindeman's studies have accentuated the sharp distinction which should be made between the basal line and the sharp elevations in the mortality curve. Contrary to their teachings I do not find that these elevations are produced by heat-strokes. They are the expression on hot days of an increase in deaths occurring in infants already fatally ill. The high basal line is a good index of the

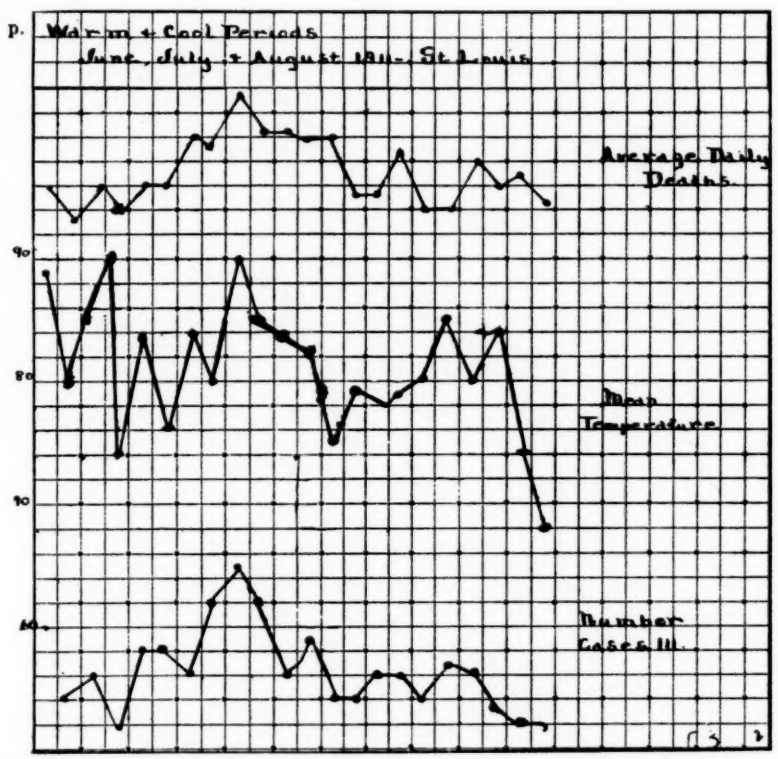

Fig. 27.—The lowest curve represents the number of cases of diarrhea beginning at each period. Taken from the St. Louis mortuary records.

morbidity. For example, in September, 1912, in spite of a marked fall in the terrestial temperature, deaths from diarrhea had not ceased, and, as proved in my private practice, new cases continually occurred. Thus in August I saw fifteen new cases, and in September also fifteen new cases, half of which were observed during the last two weeks when the temperature was low.

In fact, in St. Louis we recognize an autumn diarrhea coming on in the latter part of August and running through September and October.

These cases are characterized clinically by the passage of small, mucous stools, moderate fever, and a protracted course. The symptoms are those of an infectious colitis, and do not yield promptly to an antifermentative diet. On the other hand, the cases in July, excepting those grouped under the term of ileocolitis, exhibit a marked tendency to watery stools.

Rietschel and others have tried to find an etiologic relation between the dryness of the atmosphere and infant mortality. In fact, stress has

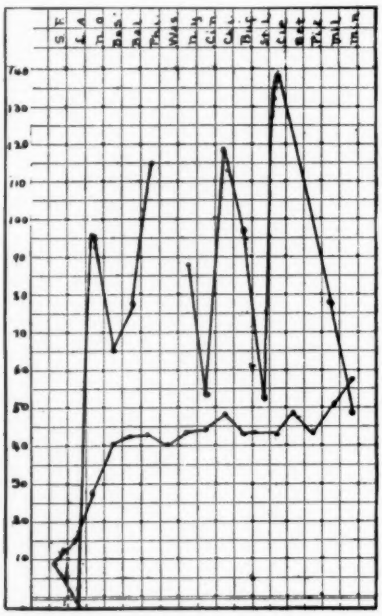

Fig. 28.—The lower curve represents the difference between the mean winter and summer temperatures; the upper curve gives the death-rate (1910) in the cities shown by the initials in the top line. It is true that the cities with the least difference show the least mortality.

been laid on the exsiccation of the tissues in the sick baby as a factor in infantile mortality. As a matter of fact, however, it is the hot humid days which cause the most cases of thermic fever.

TABLE 3.—AGES OF INFANTS DYING OF SUMMER DIARRHEA IN ST. LOUIS IN 1911 AND 1912

Age	Cases	Age Months	Cases	Age Months	Cases
1 Month or less	53	10	24	19	7
2 Months	75	11	20	20	8
3 Months	58	12	19	21	9
4 Months	60	13	18	22	3
5 Months	47	14	17	23	4
6 Months	42	15	8	24	3
7 Months	36	16	7	25	4
8 Months	34	17	7	26	3
9 Months	36	18	12	27	2

These figures emphasize the point that the second summer is not the most dangerous. Yet to the infant who has been nursed the first summer the second summer offers considerable danger. Clinical experience teaches that nearly every infant during the first and second years has one or more attacks of diarrhea. There is no disease so universal. While much stress is laid on the fact that deaths from enteritis are rare among the well-to-do, the prevalence of diarrhea among these babies does not seem to be much less than among the poor. But among the former the prompt recognition and therapy reduces the mortality to a very small figure.

While the death-rate in the country is usually less than in the cities, the babies there almost universally have diarrhea the first or second summer (personal observation).

A few additional facts may be offered on this subject.

In the summer of 1898 about 100 babies, inmates of the Bethesda Foundling Home, were taken out of the asylum and placed in an adjoining building while the asylum was fumigated for some contagious disease. This building was not screened and flies literally covered the babies all day. Two days later an epidemic of diarrhea commenced which carried off twenty-seven of the inmates in the next thirty days.

In July, 1905, all the infants were fed on raw milk of exceptional purity; nevertheless, an epidemic of dysentery occurred. More than forty infants were attacked, and fully half of them died. The disease was characterized by bloody, mucous stools and protracted fever.

At least three times I have seen severe forms of diarrhea introduced among infants in institutions by an infant from the outside sick with diarrhea.

It is useless to multiply instances or reports from other sources. We recognize in America a common form of diarrhea that has all the clinical characters of an infectious disease; that is communicable and transmitted by flies, milk and other foods; that is independent of hot days, and makes up a very large part of the deaths from summer diarrhea. To argue that heat alone by its direct effect on the organism produces the great increase in the morbidity and mortality is to deduce a general law from very few facts.

CONCLUSIONS

1. The mortality of summer diarrhea is practically the same all over the United States. The only marked exceptions are certain cities on the Western Coast.

2. The mortality of diarrheal diseases is twice as high in the summer as in the winter.

3. A room temperature of more than 85 F. has a detrimental influence on the sick baby.

4. Hyperthermia may be produced in infants when the day and night temperature is more than 85 F., but this thermic fever does not produce diarrhea nor appreciably raise the mortality rate.

5. Summer diarrhea is not the effect of high atmospheric temperature alone, but it cannot as yet be denied that excessive heat may lower the tolerance to carbohydrates and other elements of food.

6. We must seek the cause of summer diarrhea (1) in micro-organisms whose virulence and activity may be increased in the summer; (2) in endogenic or ectogenic toxic substances of unknown nature.

1460 South Grand Avenue.

THE INFLUENCE OF ATMOSPHERIC CONDITIONS ON THE MORTALITY OF INFANTS SUFFERING WITH GASTRO-INTESTINAL DISORDERS

A STUDY OF THE SUMMERS OF 1910, 1911 AND 1912 IN ST. LOUIS *

A. S. BLEYER, M.D.
ST. LOUIS, MO.

Description of Charts: The heavy upright lines in the charts (pp. 321-2-3) represent the total number of deaths from non-surgical disorders and diseases of the gastro-intestinal tract which occurred among infants in the first two years of life in St. Louis on each day of the months May, June, July, August and September of the years 1910, 1911 and 1912 (deaths in the first week of life were not included). Each horizontal space denotes one death. The numerals at the left and right margins refer to degrees of heat and relative humidity and also to average miles per hour of wind velocity.

The heavy horizontal line represents the mean of the daily maximum and minimum temperatures.

The shaded horizontal line represents the mean relative humidity for each day as determined by averaging the relative humidity at 7 a. m. and 7 p. m.

The horizontal line at base of charts represents the average wind velocity per hour for each day.

(Temperature, relative humidity and movement of air were computed from records at the U. S. Weather Bureau at St. Louis. Deaths were compiled from death certificates at the mortuary office of the St. Louis Health Department.)

As for humidity, the charts for St. Louis seem to coincide with studies conducted in other cities, notably those of Willim for Berlin and Breslau; in none could a definite relation between humidity and summer deaths among infants be shown.

The St. Louis charts show that quite often the most intimate relation between heat and deaths may exist during periods of low humidity; an instance of this may be seen in the rise of the death-curve in Chart 1 (1910), which set in about the middle of June, when the humidity was low, and remained low until the death-rate had acquired its usual summer level. This is still more strikingly seen in Chart 2, the excessive heat of 1911 continuing throughout June and July, presenting the highest death-curves of the three summers and being attended by a singularly low relative humidity. (In corroboration, it was interesting to find that curves taken in New York City during the past several summers show also that a low humidity often accompanies a high death-rate in the presence of high temperatures.) Instances illustrating the converse of this proposition are found several times in these St. Louis charts and

* Read at a meeting of the Washington University Medical Society, May 12, 1913.

show that the death-rate may be surprisingly low during excessively moist periods, even when the temperatures were high. Elevations of temperature above 71 or 72 F. were, as a rule, soon followed by a rising death-curve regardless of the presence or absence of high relative humidity.

As for the influence of heat on infant deaths from gastro-intestinal disorders, a glance at any charts of this sort will show at once that a strong relation exists; there is an obvious although imperfect parallelism between the temperature-curve and the death-curve. It was chiefly for the purpose of studying this relation that the present work was undertaken.

The nature of the influence of high temperatures on infant deaths, of those infants particularly who are suffering from gastro-intestinal disturbances, is far from clear. A view which has attracted a considerable amount of attention during the past two years is based on the assumption that heat may produce so profound an alteration of metabolism as to be followed within a few hours by death. This so-called "heat-stroke" theory was evolved not from clinical observation, but from the study of mortality charts such as are here presented; they differ from these, however, in the very salient fact that for the most part 2:00 p. m. temperatures or maximum temperatures were used, whereas in the St. Louis charts, mean temperatures for each twenty-four hours were selected.

In the case of adults, heat-stroke is clinically well known, and similar if not identical conditions have been produced experimentally in monkeys by exposing them to the sun (Aron, Philippine Islands); among infants, however, heat-stroke has not been commonly diagnosed, and if the contention made by Liefmann and Lindemann, the originators of this theory, is correct, many deaths which have been ascribed to toxic degenerations of liver or heart or to alimentary intoxications were, to be more accurate, heat-strokes.

In order to study the acute rises in deaths coincident with similarly acute rises in temperature, Liefmann and Lindemann used the terms "death-peaks" and "heat-peaks." In the present study an "heat-peak" was assumed to be the summit or climax of any ascending daily mean temperature, starting at not less than 70 degrees. During the summers 1910, 1911 and 1912 such "heat-peaks" occurred in St. Louis forty-one times; "death-peaks" coincident with these "heat-peaks" occurred in nine, 22 per cent., of instances; or, to place it the other way 'round, coincident with 78 per cent. of the "heat-peaks," the death-curve either remained stationary or dropped.

According to Finklestein, Klose and others, a more striking effect of high temperatures may be observed on the day following the summit of the heat-wave. This I found to be the case in 24 per cent. of instances; lastly, the third day was represented by 15 per cent. Further than this

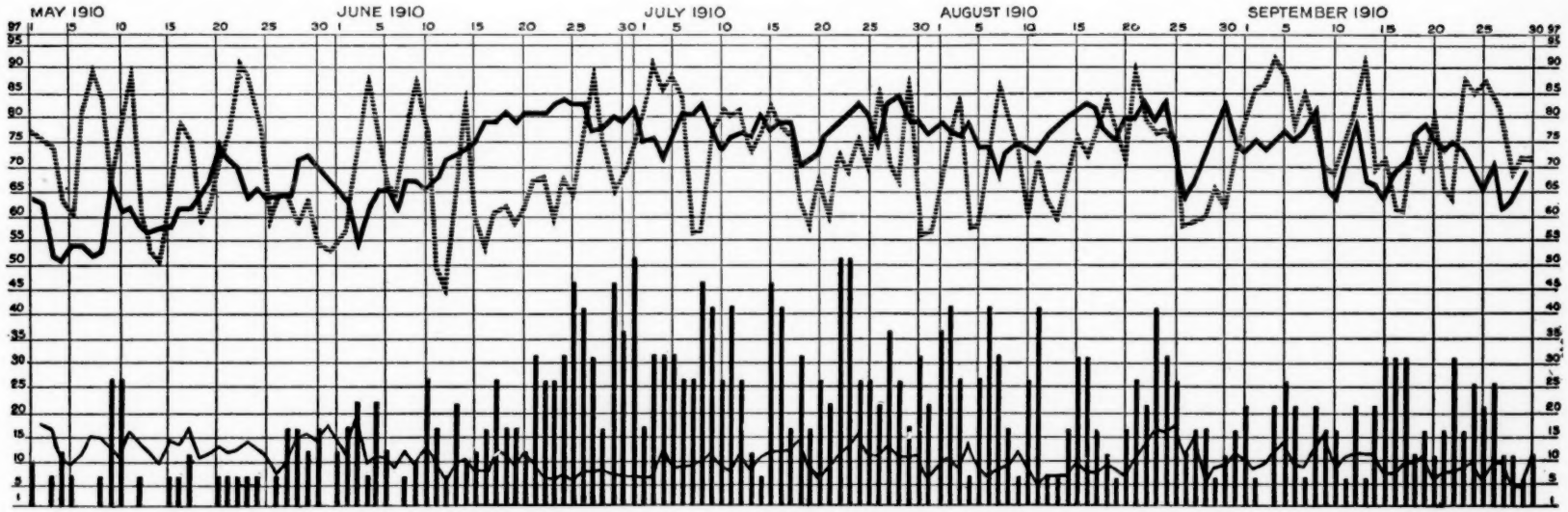

Chart 1

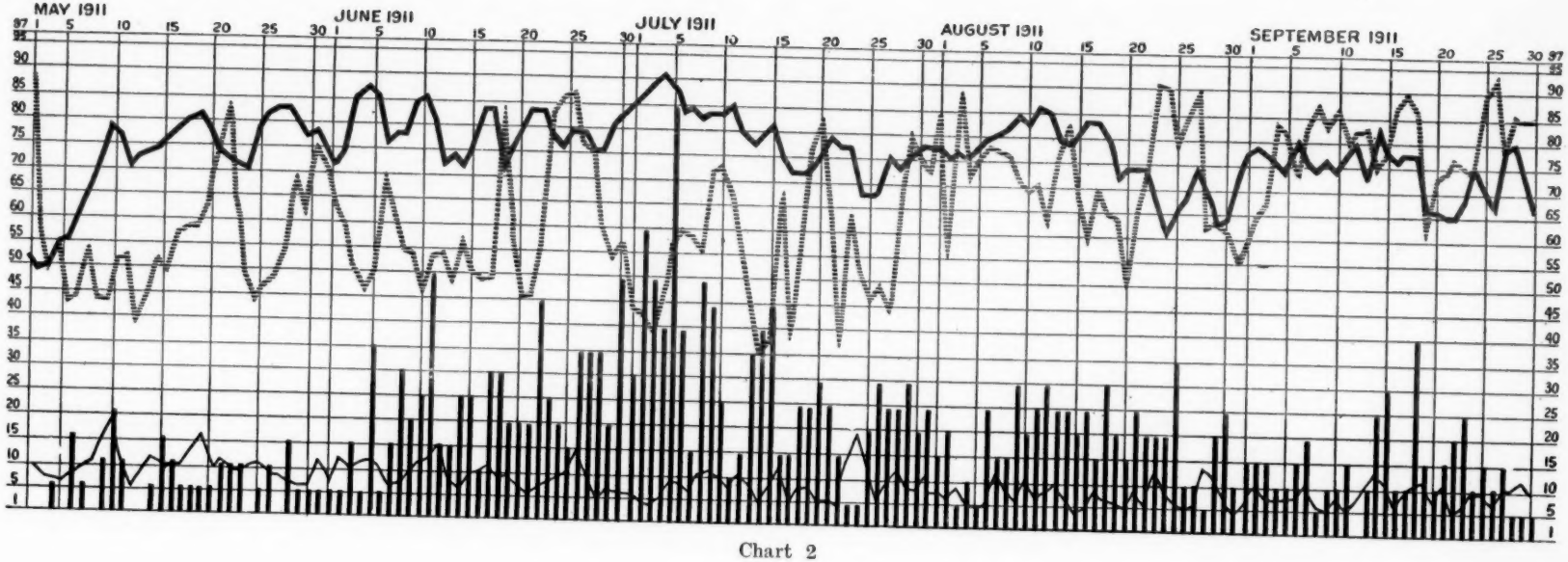

Chart 2

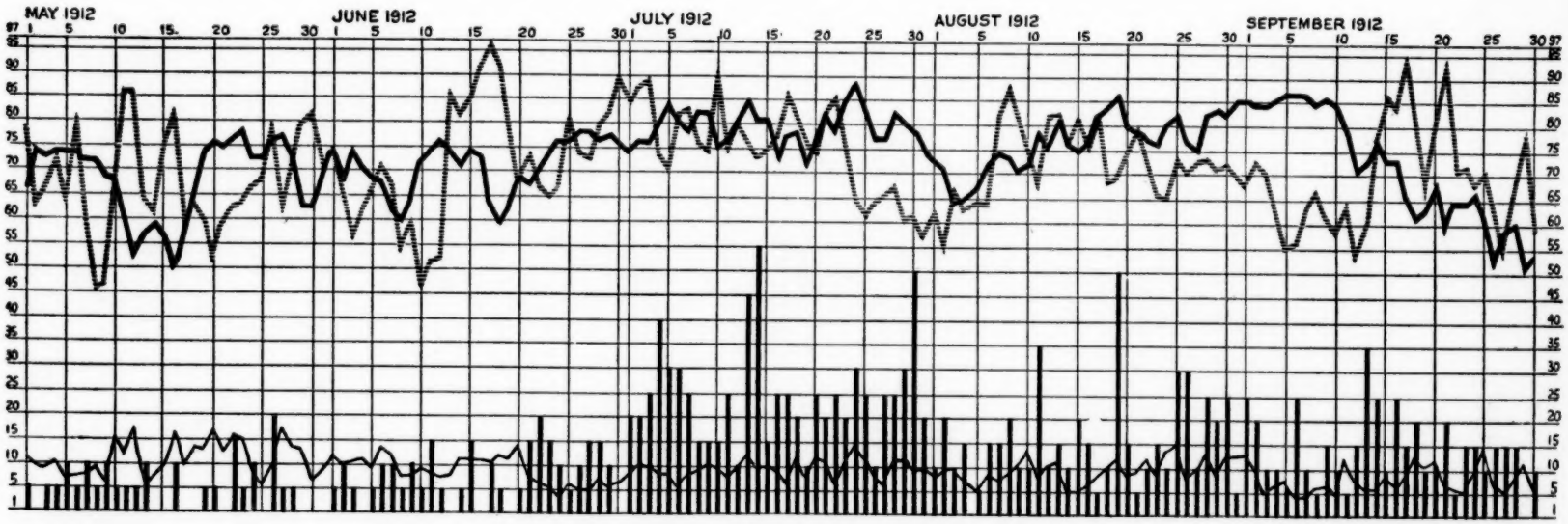

Chart 3

it was not possible to go because of the occurrence of new and conflicting "heat-peaks."

Because of these low figures it was sought to determine the behavior of the death-curve in relation to accessions of heat to points above 70 F., that is, the behavior of the death-curve was noted whenever the mean temperature reached 70, 75, 80, 85 and 90 F. This method revealed ninety-six instances in the three summers, and it was found that, corresponding with the day of the heat accession, the deaths went up in 27 per cent. of instances, that on the day after the accession it rose in 34 per cent. of instances and on the third day in 20 per cent. of instances. It is noteworthy, moreover, that the elevations of the death-curve on extremely hot days was less frequently noted than on those days when the temperature was not so high, as is seen in the following summary:

BEHAVIOR OF DEATH-CURVE IN RELATION TO ACCESSIONS OF MEAN TEMPERATURE TO 70, 75, 80 AND 85 F., SUMMARY OF NINETY-SIX INSTANCES IN 1910, 1911 AND 1912:

AT 70 DEGREES: In eighteen instances of accession to 70, the death-curve rose on the first day, four times; second day, seven times; third day, three times. In four of these eighteen instances the death-curve fell on the day the mean temperature reached 70 F.

AT 75 DEGREES: In thirty instances of accession to 75, the death-curve rose on the first day, eleven times; second day, eight times; third day, four times. In five of these thirty instances the death-curve fell on the day the mean temperature reached 75 F., and in two instances it remained stationary.

AT 80 DEGREES: In thirty instances of accession to 80, the death-curve rose on the first day, eight times; second day, thirteen times; third day, eight times. In six of these thirty-six instances the death-curve fell on the day the mean temperature reached 80 F., and in one instance it remained stationary.

AT 85 DEGREES: In twelve instances of accession to 85, the death-curve rose on the first day, three times; second day, four times; third day, four times. In one of these twelve instances the death-curve fell on the day the mean temperature reached 85 F.

In ninety-six instances the death-curve dropped or remained stationary in twenty instances; the rise of the death-curve on the day of the accession of heat was more frequent at 75 F., than at 80 F., or 85 F.

In studying the relation of heat to deaths, the originators of the heat-stroke theory found this relation to be most evident in the early summer months. Some of the German charts show this very nicely, but the charts for St. Louis do not show so great an intimacy between heat and deaths in the early months as they do in the later months of the summer; for example, in 1910, the first death-rise in May is not accompanied by a coincident rise in temperature, and a fall in temperature precedes the death-rise of May 27-30. Again, the rising death-curves of early June accompany a really low temperature, but were preceded by moderate heat a week earlier. The fastigium of heat of middle and late June is followed rather than attended by the rising deaths, and such relations may also be seen, although to a less definite degree, elsewhere in

the chart for this year. In the 1911 chart it may be noted that the deaths follow rather than accompany the excessive heat of June and July, although it had already been so extremely hot for so long a time that the curves become confused. Lastly, the rise in deaths in late September accompany a lowered temperature curve, but follow a higher preceding one. In 1912, the deaths of late June are apparently more closely related to the heat of middle June than to that of late June, and this is again seen in the rise of late September in the presence of very low temperatures, following, however, the very high temperatures earlier in the month. It would appear that the death-curve exhibits a sequential rather than an accompanying rise to the heat-curve, the hot days often passing without leaving an immediate impress in deaths.

As for the relation of wind velocity to infant mortality in summer, the subject may be dismissed at this time with a word — no relation could be made out. This does not mean that a relation does not exist, since to the contrary, it has been shown that movement of air is capable of prolonging the lives of animals exposed to the sun; but the charts do not show this.

CONCLUSIONS

A definite relation between atmospheric heat and deaths among infants suffering with gastro-intestinal disturbance exists; at what degree of heat lethal effects are seen does not appear in these charts.

A. So-called "heat-peaks" or short-lasting but excessive temperatures doubtless emphasize, but they do not dominate the summer infant death-rate; whether many of these deaths are the direct result of "heat-stroke," although easily supposable, has not been shown.

B. Continuous heat shows a stronger relation, although not necessarily an immediate relation, to deaths of infants suffering with gastro-intestinal disorders, which, as infants are now housed and fed, is very fairly constant.

I wish to thank Mr. Montrose Hayes, Chief of Weather Bureau at St. Louis, for assistance in compiling material for the charts.

Delmar Building.

THE COMPLEMENT FIXATION REACTION OF THE BLOOD OF CHILDREN AND INFANTS, USING THE BACILLUS ABORTUS AS ANTIGEN *

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MINNEAPOLIS

The passage of substances through the intestinal wall, and the effect of the ingestion of biologically active bodies on the reactions in the blood and other tissues has been a subject of much interest to many, and especially so to pediatrists. We hope to throw some light on this problem by the demonstration of the fact that a specific reaction can be obtained in the blood of a considerable number of children.

The specific reaction in question is that of the complement fixation test, using the bacillus of infectious abortion of cattle as an antigen. The significance of this particular reaction becomes apparent when we consider that epidemic abortion is common among cattle; that the presence of the *Bacillus abortus* can be demonstrated in cow's milk, and that the reaction is seemingly specific.

The discussion concerning the enteral absorption of homologous or heterologous proteins has been, and still is, of great interest, but if we turn at once to the subject of the effect of the ingestion of biologically active substances, such as toxins and antitoxins joined with proteins, several illuminating studies are at our disposal.

Römer[1] was able to show in 1901 that a colt, the blood of which at birth was found to be antitoxin-free, and which was fed with its mother's milk which contained diphtheria antitoxin, then gave proof of having antitoxin in its own blood. After the twelfth day, however, no further antitoxin absorption could be demonstrated, and the antitoxin content of the colt's serum then rapidly decreased.

This is of especial interest in connection with the position taken by von Behring concerning other proteins and the tuberculosis question, suggesting the greater possibility of absorption of such products by the new-born.

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* Read before the Chicago Pediatric Society, April 21, 1913, and before the American Association of Medical Milk Commissions, at the Seventh Annual Meeting, held at Minneapolis, June, 1913.

* Attention is directed to the paper of Dr. E. C. Schroeder in this issue containing comments on *Bacillus abortus*.

1. Römer: Untersuchungen über die intrauterine und extrauterine Antitoxinübertragung von der Mutter auf ihre Deszendenten, Berl. klin. Wchnschr., 1901, xxxviii, 1150.

Salge[2] gave antitoxic horse serum by mouth to human infants. In no instance was he able to demonstrate the passage of the antitoxin into the infants' blood, even in the new-born. When, however, the antitoxic horse serum was injected subcutaneously into the mother or nurse, and the child put to the breast, the passage of the antitoxin to the child's blood could be shown.

Uffenheimer experimented with the administration of serum-hemolysin, casein, egg-albumin, diphtheria and tetanus antitoxins, by mouth, to new-born guinea-pigs. The hemolysin and casein did not pass over. The egg-albumin could be demonstrated in traces only. The antitoxins could, however, be demonstrated in the blood of the new-born pigs.

The recent work of Hehn taken with that of Lust[3] indicates that antitoxins in heterologous serums are absorbed more readily in certain diseased conditions of the alimentary tract.

As the infectious abortion of cattle is a condition which, although well known to veterinarians, has but recently appeared in medical literature, a discussion of the condition may be of value.

The *Bacillus abortus,* often referred to in the literature as the cocco-bacillus of Bang, was discovered in 1896 by Bang and Stribolt of Copenhagen.

Epizootic abortion has for the past century been the greatest curse with which stock-breeders and dairymen have had to contend. For nearly one century scientific men have recognized the contagious character of this disease, but thus far no advance has been made by way of combating contagious abortion either prophylactically or therapeutically.

With the dawn of the bacteriologic era began the hunt for the etiologic agent of contagious abortion. Men so eminent as Nocard worked on the problem not less than three years without being able to throw any light on its etiology.

The discovery of Bang[4] and Stribolt was soon confirmed by Stockman and McFadyean[5] of Great Britain and Nowak[6] of Austria, and more recently the cocco-bacillus of Bang has been found in this country.

BACILLUS ABORTUS BANG

Bang's bacillus belongs to the smallest of the visible micro-organisms, measuring from 1 to 2 microns in length. It is a non-motile, non-spore-bearing cocco-bacillus, one diameter being only slightly greater than the

2. Salge: Immunisierung durch Milch, Jahrb. f. Kinderh., 1905, lxi, 486.
3. Lust: Die Durchlässigkeit des Magendarmkanals für heterologes Eiweiss bei ernährunggestörten Säuglingen, Jahrb. f. Kinderh., 1913, lxxvii, 243.
4. Bang: Ztschr. f. Tiermed., 1897, i;Maanedskrift f. Dyrlaeger, 1900.
5. McFadyean and Stockman: Report of Committee to Inquire Into Epizootic Abortion, London, 1909.
6. Nowak: Ann. de. l'Inst. Pasteur., 1908, xxii, 541.

other. It may readily be stained by any of the ordinary anilin dyes, but is Gram-negative.

The micro-organism in question will grow on any of the ordinary culture media after it has once been cultivated in the laboratory. Bang and Stribolt noticed that the growth of the *Bacillus abortus* appeared a few millimeters beneath the surface of the culture medium and hence concluded that it was a semi-aerobe, developing only at such a point as presented a given oxygen pressure. These authors made the further observation that after this micro-organism had once been cultivated in the laboratory succeeding generations thrived best in an atmosphere of nearly pure oxygen.

McFadyean and Stockman found that the *Bacillus abortus* could be cultivated successfully when grown in symbiosis with the *Bacillus subtilis*. These authors assumed that the *B. subtilis* by absorbing a part of the oxygen contained in the closed chamber created a favorable atmosphere for the development of the *B. abortus*. If the observations of these European authors are correct relative to the *B. abortus* in its relation to oxygen, we have here a phenomenon quite unique. It seems strange that an organism which in the first generation develops only under conditions of diminished oxygen pressure should swing to the opposite extreme in the succeeding generation.

There is another possible explanation of this, however.

All who have worked with this organism will agree that the growth of the first generation on artificial media is slow. The colonies, as a rule, do not appear until at the end of the fourth or fifth day, and sometimes even eight to ten days may elapse before the growth becomes apparent. During this time the surface of the culture medium has a tendency to dry out and the growth appears deeper probably because of its demand for moisture.

We have further found that the *B. abortus* grows very well on the surface of ordinary slant agar provided the tube is well sealed with paraffin to prevent evaporation. In a previous paper[7] we have expressed the view that this *might* be explained by the fact that agar absorbed a part of the oxygen contained in the tube, thus creating a favorable environment for the development of the *B. abortus*.

When Bang's bacillus has once been cultivated in the laboratory it grows readily on any of the ordinary media.

The isolation of the *B. abortus* presents but few difficulties. It will, as a rule, be found in pure culture in the gastro-intestinal tract of the fetus provided this is obtained within a few hours after it has been expelled.

7. Larson, W. P.: Jour. Infect. Dis., 1912, x, 178.

TECHNIC OF CULTIVATION OF B. ABORTUS

The technic we employ is to inoculate tubes of slant agar, preferably ascitic agar or serum agar, from the gastro-intestinal tract of the fetus, and either seal the tubes with paraffin or place in a closed flask together with a few tubes of slant agar freshly inoculated with the *B. subtilis.* On the fourth or fifth day, occasionally on the third day, the growth on the surface of the medium is apparent in the form of small dewdrop-like colonies, which at first remain isolated, but in the course of the next few days become confluent. The *B. abortus* does not coagulate milk nor liquefy gelatin. It produces neither acid nor gas on any of the sugar media. On potato it resembles very much the *B. malleus.*

It has until quite recently been held that the bacillus of Bang was not pathogenic for any of the laboratory animals, with the possible exception of mice. The work of Smith and Fabyean[8] and Schroeder and Cotton[9] shows that this organism is pathogenic for guinea-pigs. Injected intravenously into pregnant rabbits or guinea-pigs it will often interrupt pregnancy.

Smith and Fabyean[8] have recently found that when injected into the peritoneal cavity of guinea-pigs the bacillus of Bang produces lesions very similar to those of tuberculosis. They report observations made on fifty-eight guinea-pigs and they found lesions in the following organs: lymph-nodes, spleen, liver, kidney, testicle, lung, bones. The eyes of some of these animals presented lesions in the form of an opacity of the cornea.

Fabyean[10] found further that the guinea-pigs thus affected were hypersensitive to a toxin extracted from the cultures of the *B. abortus,* even when injected in small doses, causing the death of the animals in from twelve to forty-eight hours, thus resembling the action of tuberculin when injected into tuberculous animals.

In the summer of 1911 we became interested in contagious abortion of cattle and inaugurated studies of the disease as it affects bovines. Our work was directed mainly toward establishing the identity of the American and European diseases by the complement-fixation method.

Wall[11] and Holt,[12] working in the laboratory of C. O. Jensen of Copenhagen, had found that contagious abortion of cattle could be accurately diagnosed by this method. We were able to confirm their work. We found that antigen prepared from cultures of the *B. abortus* received from Denmark fixed the complement in the presence of serum

8. Smith and Fabyean: Centralbl. f. Bakteriol., lxi, 549.
9. Schroeder and Cotton: Leaflet issued from the Bureau of Animal Industry, 1912.
10. Fabyean: Jour. Med. Research, xxi, 441.
11. Wall: Maanedskrift f. Dyrlaeger, 1910, xxi.
12. Holt: Maanedskrift f. Dyrlaeger, 1911, xxii, 1.

from an infected animal. Cultures which we isolated from infected material in this country were identified with the Danish organism.

We have, during the past two years, had occasion to examine the blood of several hundred animals and have found the complement-fixation method to be a very accurate means of diagnosing this disease.

Epizootic abortion is probably the most prevalent of all animal diseases. There is scarcely a dairy community where the disease has not at some time prevailed. Young heifers of the herd are the first to become infected and abort. Once introduced into a herd it passes from animal to animal until nearly the entire herd has become infected. An animal will usually abort two to three years in succession, after which she is apparently immune, and when not made sterile by the disease, will carry to term. The abortions usually occur from the third to the seventh month of gestation. The general health of the animal is apparently undisturbed by an infection with the Bang bacillus.

It is generally supposed that the male element of the herd transmits the disease from animal to animal, but there is at present no definite proof of this. Cultures made from bulls of infected herds have invariably been negative; likewise blood examinations made on some fifty bulls have all given negative results.

Bang was able to cause cattle, sheep and goats to abort by administering cultures of the organism per os.

Owing to the prevalence of the disease one would naturally expect to find the organism in milk.

In a study of the pathogenic organisms found in milk, Schroeder and Cotton accidentally found the *B. abortus,* but for some time were not able to identify it. They injected milk as obtained on the market into the peritoneal cavity of guinea-pigs and not infrequently found tubercle-like lesions of the abdominal viscera following such injections in about 10 per cent. of the animals thus treated. They finally concluded that the lesions were due to the *B. abortus.*

It has been pretty conclusively demonstrated that all of our domestic animals are susceptible to infection by this organism, although cattle are by far the most frequently affected. Bang found that sheep and goats would abort after being experimentally infected with this organism. Ostertag[13] isolated the organism from mares which had aborted. The usual laboratory animals are likewise susceptible to this disease. About one year ago we were forced to discontinue our complement-fixation work on this disease owing to the fact that fully 60 per cent of our guinea-pigs gave a positive reaction, in consequence of which their serum could not be utilized as complement.

13. Ostertag: Kolle and Wassermann's Handbuch d. path. Mik.

HUMAN INFECTION

While we were making field observations we took particular pains to make inquiries as to whether there had occurred abortions in women who had come in contact with a herd of cattle while an epidemic of contagious abortion prevailed. Such observations would naturally be of but little value unless one had the opportunity of observing a large number of cases. In the limited number we have had occasion to observe, we have recorded cases of abortion of the mother of the household where the ordinary causes, such as lues or injuries, could be definitely excluded. The results of our investigation along this line will be presented in another paper. Suffice it to say that in systematically examining the serum of women who have aborted we have found that a larger number give a positive complement-fixation reaction using the *B. abortus* as antigen than when the ordinary syphilitic antigen is used.

Thus having evidence indicating that humans might become infected with this organism, we proposed to carry our investigation further in the hope of determining whether or not and to what extent the micro-organism in question might be of pathological significance to the human family.

As indicated elsewhere in this paper, the bacillus of Bang is somewhat difficult to cultivate, especially when found in impure culture. In view of this fact the ordinary bacteriological procedures did not appeal to us as being the most desirable as exploratory measures; hence we then proposed to push our investigations further by means of the complement-fixation reaction.

From the work of Cotton and Schroeder we know that 10 per cent. of market milk contains the *Bacillus abortus;* there can, therefore, be little doubt that any individual using dairy products is amply exposed to infection. Bang has shown that animals may readily be experimentally infected with this organism by administering it per os. If the *B. abortus* is pathogenic for human beings, as it is for guinea-pigs, and can be transmitted through the digestive tract as is the case with cattle, we would expect infants and children to be the subjects most frequently infected. We have to date examined the blood of 425 children by the complement-fixation method and have found at least some support of the above-expressed hypothesis. Of these 425 cases we found seventy-three positive and 352 negative reactions. In other words, the blood of 17 per cent. contained antibodies against the *B. abortus.* The same technic was used as in the examination of bovine serums. In order to guard against every possible source of error, known positive and known negative bovine serums were invariably used as controls. Our work was not confined to the complement-fixation test, but agglutination tests were made in a large number of cases. We found in working with human serum, as did Holt

with bovine serum, that the agglutination and complement-fixation tests run parallel.

To further corroborate the evidence that we were dealing with specific antibodies, other methods suggested themselves. In their work on hemolysis, Ehrlich and Morgenroth found that the specific hemolytic antibodies would unite with the washed erythrocytes when the latter were placed in the serum and left for a few hours. By centrifuging this mixture and decanting off the supernatant serum they found that the latter had, by the contact with the blood-cells, been depleted of the hemolytic antibodies. In other words, the antibodies had attached themselves to the blood-cells and been removed with these.

The same is true of the *B. abortus* antibodies. By treating a positive serum with a suspension of the Bang bacillus, the serum, after a few hours' contact, has been found to be negative when separated from the organisms by the centrifuge. If an adequate amount of complement is then added to the sensitized bacilli and incubated for an hour, the complement will become fixed, thus indicating further that we are dealing with specific antibodies.

It behooved us to prove that a positive serum could not be rendered negative through the action of various indifferent bacteria, such as members of the colon typhoid group, the proteus and pyogenic bacteria. During the course of this investigation a large number of positive serums have been treated with one or more of the above-named bacteria, but invariably with a negative result.

The one question which presents itself is: Are these antibodies in the blood of children the result of an active or passive immunity? This question we have found difficult to answer satisfactorily. We recall the experiments of Ehrlich[14] in which he immunized mice against rizin and abrin, and later gave the young of non-immunized animals to the immune animals and *vice versa;* he found that the young of non-immune mothers would become immunized against these poisons when allowed to suck the immune animals for a time. If the same law applies to the problem we now have under consideration, we could readily explain our positive reactions on another basis than infection of the individual.

The experiments of Ehrlich show very clearly that immune bodies are excreted in the milk and that these may pass through the mucous membrane of the intestine and reach the blood. Much and Römer, in studying this phenomenon, further conclude that the penetrability of the mucous membrane of the new-born for immune bodies is by far greater than that of adults. These authors found that the source of the milk containing the immune bodies was no insignificant element. The anti-

14. Ehrlich: Ztschr. f. Hyg., 1892, xii.

bodies contained in milk of a homologous species were absorbed much more readily than when heterologous milk was given.

Attempts were made to demonstrate the *B. abortus* antibodies in milk from cows which had recently aborted, and whose blood serum fixed the complement in high dilutions. Our results have thus far been negative, as large quantities of milk interfere with the reaction.

Attempts to deplete the milk of any eventual antibodies by treating it with an emulsion of the Bang bacillus have likewise failed to give satisfactory positive evidence that such exist in milk. It is very probable that our positive reactions were the result of an active immunity; in other words, due to antibodies generated by the individual examined. It does not necessarily follow that the positive reactions were due to an active infection of the individual. The work of Müller and others indicates that it is possible to immunize an animal by introducing the antigen per os or per rectum, provided large and repeated doses of the antigen are administered.

IMPORTANCE OF HUMAN SUSCEPTIBILITY

This discovery of so common a reaction in children opens up the opportunity for more study. We have first the problem as to whether this is a passive or active immunity. Then comes the question concerning the process of absorption, together with further theoretical considerations.

To the pediatrist, it is of particular interest that inoculations of guinea-pigs with the *B. abortus* has been shown to produce lesions of the lymph-nodes, spleen, liver, kidney, testicle, lung and bones, and that these lesions are suggestive, anatomically, of those of tuberculosis. These findings of competent observers together with the experience that inoculation may produce epiphyseal enlargements, and our 17 per cent. of positive reactions among children with anatomical lesions of the osseous system, most of them with clinical diagnoses of tuberculosis and rickets, indicate very clearly the need of further investigation.

AN EXPERIMENT WITH RAW AND HEATED COW'S MILK AND ITS LESSON. WITH COMMENTS ON BACILLUS ABORTUS *

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In January, 1910, my assistant Dr. George W. Brett and I undertook an investigation on the relative value of raw, pasteurized and boiled cow's milk as a food for unweaned animals. Experimental studies on this subject commonly have one or both of two weaknesses — the use of an insufficient number of animals and an inadequate period of time during which observations are made; hence Dr. Brett and I determined to use the largest number of animals we could properly handle and keep them under observation in all cases from birth until they died or reached adult life, and in some cases after they had reached adult life, to test their vital efficiency as far as this would be measurable by the number and vigor of their progeny during one year.

The animals selected for our investigation were guinea-pigs, as they were the only species of which a sufficient number of the proper age and a definitely known history could be obtained. We realized that data derived from guinea-pigs could not be applied, off-hand, to all species of animals that are at times fed on cow's milk under artificial conditions as a substitute for mother's milk under natural conditions, but we assumed that they would have a fairly broad significance, especially as the main argument against the use of heat to destroy those disease germs with which ordinary commercial milk is too often contaminated, and which cannot be excluded unfailingly from milk that is produced with unusual precautions under exceptionally favorable conditions, is that milk contains important thermolabile enzymes and that its proteins and lime salts are modified by heat in a way that reduces their digestibility and food value.

If heat reduces the quality or the value of the milk provided by Nature for the young of cattle, it is not unreasonable to assume that this can be shown as well or better by tests with guinea-pigs, or animals that require food much like that of cattle, as with animals that are designed to digest food of a wholly different kind. The assumption, on the other hand, that cow's milk includes anything in its physical or chemical

* Attention is called to the paper of Larson and Sedgewick in this issue on the *Bacillus abortus*.

character, thermolabile or thermostabile, that has more value for infants than for the young of cattle or other truly vegetarian species of animals, on general biological principles, is hardly tenable, as Nature cannot reasonably be supposed to provide through the mothers of one species of animals for the needs of the progeny of another wholly unrelated and distinct species.

From one point of view guinea-pigs are not ideal animals for an investigation of the kind we made, and that is their remarkable development at birth and the short period of time during which they are dependent on their mothers for sustenance. But in this respect calves and young guinea-pigs, if we take the respective sizes and lengths of life of cattle and guinea-pigs into consideration, are much alike. Both species are born with their eyes open and their bodies well coated with hair; both are able to move about with ease on their own feet a few hours after birth, and neither has a long period during which milk is an essential article of food. Guinea-pigs are weaned when they are about 20 days old, and calves when they are from 5 to 8 weeks old. Hence, calves and guinea-pigs should be benefited more nearly alike by anything of a thermolabile character in cow's milk, or should suffer more nearly alike from changes induced in cow's milk through its exposure to heat, than calves and infants, or than calves and young animals that are less developed at birth and longer dependent on a milk diet.

The guinea-pigs used in the investigation, a total of 467 that were artificially fed on cow's milk, were removed from their mothers immediately after they were born and divided into three groups as follows:

Group 1, 155 guinea pigs, fed raw milk.
Group 2, 156 guinea pigs, fed pasteurized milk.
Group 3, 156 guinea pigs, fed boiled milk.

As this large number of new-born animals could not be secured or properly handled at one time, notwithstanding that the guinea-pig breeding stock at the Experiment Station is abundant and prolific, the actual, artificial feeding of young guinea-pigs extended over a period of ten months, from January to October, inclusive. Each time new-born animals were selected from the breeding pens they were divided into three equal lots, and one lot added to each of the three previously designated groups.

The milk for the tests was obtained from Experiment Station tuberculin-tested cows; in quality it was equal to or better than the best milk obtainable at any price in our cities, and none of it was more than eight hours old at the time it was fed to the guinea-pigs. The pasteurized milk was exposed to a temperature of 60 C. (140 F.) for twenty minutes and then rapidly cooled, and the boiled milk was slowly heated, in about

ten minutes, to the boiling point, and kept at that temperature one minute and then rapidly cooled.

During the first twenty days of their lives the guinea-pigs were fed five times daily with a special nursing apparatus, consisting of a graduated glass tube fastened to a wooden stand by means of screw clamps and communicating through a fine rubber tube with a nipple. The little animals quickly learned the use of this apparatus and showed as much affection for it as an artificially fed infant shows for its bottle.

The amount of milk given at each feeding was 1 cubic centimeter. After the twentieth day a pan of milk was accessible to the guinea-pigs for ten days longer, and then all milk was withdrawn from their diet. From the beginning the animals had access to other food than milk, and most of them began to eat small quantities when they were only a few days old.

An attempt was made to do the same work with rabbits, but the death-rate among them during the first ten days was so high that it was abandoned. Even when the young rabbits, which are born with their eyes closed and their bodies naked, were kept in a soft nest in an incubator after removal from their mothers, most of them died before they were a week old.

The difference in the value of the three kinds of milk, raw, pasteurized and boiled, was measured in two ways — by the mortality among the experimental animals and by the average weight at different periods of the animals that remained alive. The accompanying table gives the mortality record.

TABLE 1.—PERCENTAGE MORTALITY AMONG THE GUINEA-PIGS

Mortality	Young with Mothers, Per Cent.	Young Fed Artificially on Cow's Milk		
		Raw Per Cent.	Pasteurized, Per Cent.	Boiled Per Cent.
First ten days.....	4.0	27.74	30.13	27.56
First twenty days..	5.0	40.00	46.16	39.74
First thirty days...	6.0	44.52	51.28	42.95
First year	14.0	52.90	53.85	46.79

The first column of percentages is based on guinea-pigs that were raised by their mothers in the Experiment Station breeding pens, from which all the animals for the investigation were derived.

The enormous difference in this table between the death-rates of the guinea-pigs that remained with their mothers and those that were fed artificially on cow's milk, or the milk of a foreign species, is very impressive. The percentage death-rate for the guinea-pigs left with their

mothers is based on all the young born alive, weaklings as well as others, and no weaklings were selected for the artificial feeding tests. If we bear this in mind, the difference between the use of mother's milk under natural, and of the foreign milk under artificial, conditions will be recognized as really greater than the figures in the table show it to be.

The difference between the results with the raw, pasteurized and boiled milk are in favor of the boiled milk. During the first thirty days of life there is no well-marked difference between the raw and boiled milk, and both, unexpectedly, have a better record than the pasteurized, which latter, however, seems to have left the animals alive at the end of thirty days in such condition that a sufficient proportion of them

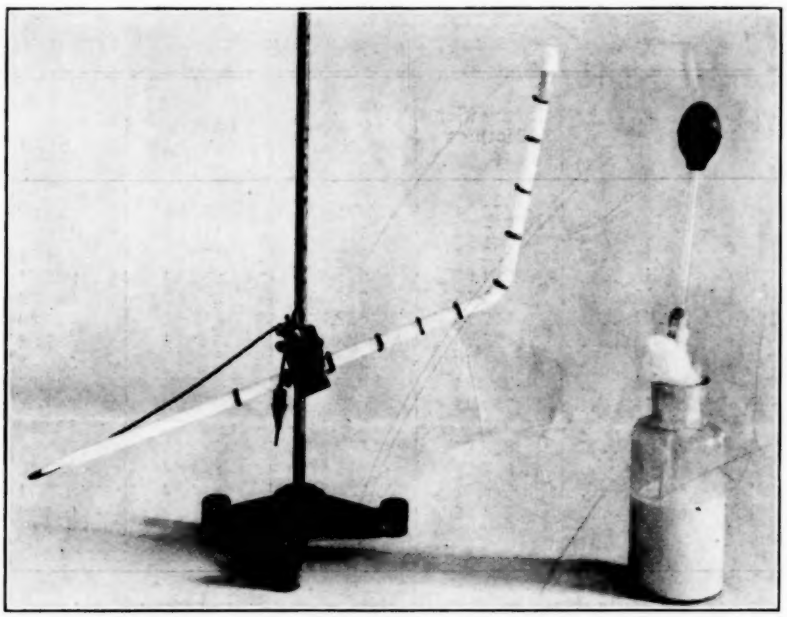

Fig. 1.—Apparatus with which young guinea-pigs were artificially fed on cow's milk.

remained alive during the following eleven months to make the records for the raw and pasteurized milk at the end of the year practically alike.

The percentage mortality table, reduced to its simplest terms, means that, in four groups of new-born guinea-pigs, each of which was composed of 156 animals, 134 of the natural group were alive at the end of the year, while of the other three groups, only 74 raw, 72 pasteurized and 83 boiled cow's milk animals survived. The natural group is 51 animals, equal to nearly one-third of the total number in each group, ahead of the boiled cow's milk group, which latter is 9 animals ahead of the raw and 11 ahead of the pasteurized cow's milk groups.

The better showing of the boiled milk must be considered with a clear appreciation of the fact that we are not dealing with ordinary commercial milk, but, on the contrary, with a milk of superlative quality.

The causes of death were mainly inflammation of the stomach and bowels and pneumonia. The post mortem examination of some of the animals that died very young revealed no satisfactory lesions to account for death.

The next table (Table 2) shows the average weight of the guinea-pigs in each group at different ages.

The first column of Table 2 is based on the average weight of 100 guinea-pigs raised under normal conditions in the Experiment Station breeding pens.

TABLE 2.—AVERAGE WEIGHT IN GRAMS OF GUINEA-PIGS AT DIFFERENT AGES

Time of Weighing	Young with Mothers	Young Fed Raw Cow's Milk	Young Fed Pasteurized Cow's Milk	Young Fed Boiled Cow's Milk
At birth	75.94	75.94	76.04	76.22
10th day of life.....	127.34	116.27	118.37	119.35
20th day of life.....	182.84	175.19	176.68	180.57
30th day of life.....	234.65	228.81	230.04	233.71
50th day of life.....	349.15	317.25	320.23	318.36
80th day of life.....	502.29	422.16	427.17	430.49
110th day of life.....	597.76	528.30	523.84	531.88
140th day of life.....	671.56	587.81	596.72	592.43
170th day of life.....	732.34	645.29	655.69	646.07
200th day of life.....	776.63	697.12	700.15	699.14
230th day of life.....	822.00	741.14	741.65	750.58
260th day of life.....	858.84	789.48	781.61	795.42
290th day of life.....	913.27	817.87	813.08	839.46
320th day of life.....	944.07	853.81	836.15	879.71
350th day of life.....	965.23	885.03	872.99	904.86
End of first year....	974.29	895.51	888.40	920.86

It is remarkable how nearly the average weights of the guinea-pigs in the different groups correspond on the thirtieth day, or ten days after weaning. This is partly due to the fact that the weaker and less thrifty animals in the artificially fed groups had been practically all eliminated by death before they were thirty days old, and that a much larger proportion of the weaker and less thrifty remained alive under natural conditions. After the thirtieth day the average weight in the natural group increased more rapidly than in the artificial groups, and at the end of the year shows a clear advantage of 53.5 gm. over the heaviest artificial group.

As in the mortality records, the boiled milk weight record is distinctly better than that of either the raw or the pasteurized milk, and the pasteurized drops a little behind the raw milk.

It is not fair to base conclusions on such slight differences as we have in this investigation, both as to mortality and weight, between the raw and pasteurized milk animals, else one might be inclined to infer, with the better records for the boiled milk, that the milk of a foreign species, when it is used as food for unweaned animals, contains both desirable and objectionable thermolabile elements, and that the desirable elements, which we can well afford to sacrifice to be rid of the objectionable, are

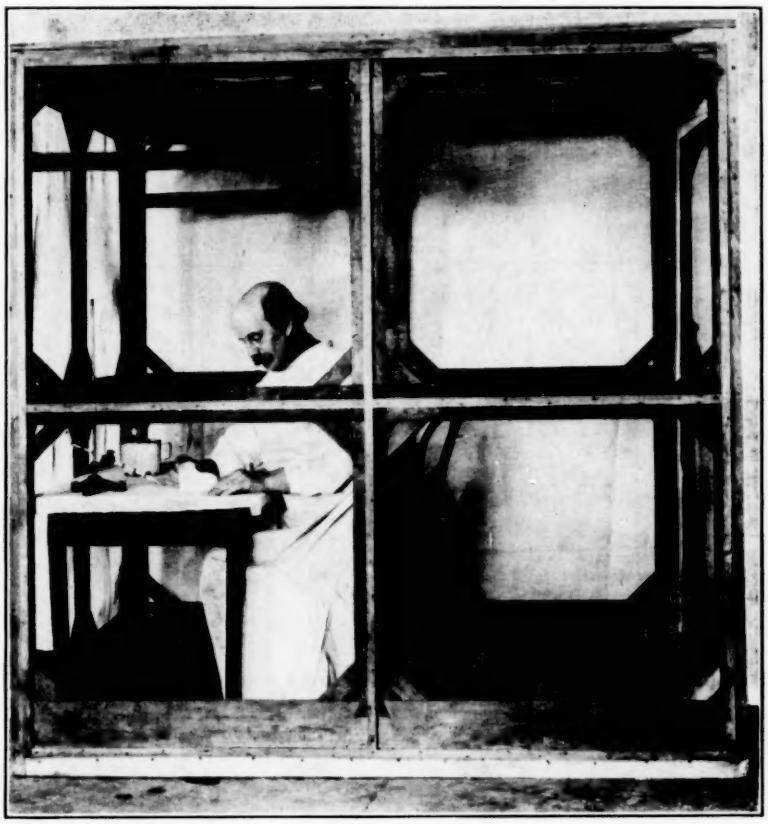

Fig. 2.—Young guinea-pig being fed artificially on cow's milk. The feeding was all done in fly-proof cages.

affected by lower degrees of heat to a greater extent than the latter. If the difference between the raw and the pasteurized milk was as great as the difference between the raw and the boiled, we would have satisfactory grounds for this inference. Incidentally, I would like to say that there are problems along this line regarding which we are very much in the dark, and that, apart from any treatment artificially used milk may require because of its contamination with pathogenic or other micro-

organisms, we have just as much evidence to support a hypothesis to the end that the milk of one species should be neutralized by heat before it is used as food for the unweaned young of another species, as to support the more commonly entertained hypothesis that the milk of one species contains thermolabile properties that are valuable for the young of another species.

From the guinea-pigs that were alive and well when they reached the age of 1 year, a number of males and females, fair representatives of each of the different groups, were selected and placed under conditions identical with those under which the Station's regular breeding stock is kept, and this gave us the data on which Table 3 is based.

The young were kept under observation until they were 30 days old, at which age there was no material difference among those from different groups of parents.

TABLE 3.—Breeding Record

Guinea-Pigs Raised on	No. of Males	No. of Females	No. of Litters	No. of Young	Young Born Dead	Young Living Less Than 30 Days	Young Alive at End of 30 Days
Mother's milk	5	15	62	210	12	11	187
Raw cow's milk.....	5	15	37	120	15	6	99
Pasteurized cow's milk	5	15	52	170	15	8	147
Boiled cow's milk.....	5	15	52	177	11	10	156

Table 3 shows clearly that the artificial feeding of young animals on the milk of a foreign species imposes a handicap which may persist long after the milk-drinking period of life has been left behind. The naturally raised guinea-pigs show a greater productivity than the artificially raised, and again the boiled milk is more satisfactory than the raw or the pasteurized.

For the very poor record of the raw milk at this stage of the investigation, however, a fairly satisfactory explanation has been found, and this is based on the fact, discovered by Dr. W. E. Cotton and myself, that the bacillus of infectious abortion of cattle commonly occurs in the milk of infected, apparently healthy cows that produce healthy calves at full term.

While milk was being fed to our guinea-pigs it was not known that one cow in the herd that supplied the milk was infected with the abortion bacillus, and I do not see how we could have known, or discovered, or guarded against, this condition. At the time it was not known that the abortion bacillus of cattle causes peculiar, almost unmistakable lesions in guinea-pigs, which develop slowly, rarely cause death and gradually

disappear. Neither was it known that the bacillus is eliminated from the bodies of infected cows with their milk, as it had never been demonstrated to occur in milk. Cotton and I had previously discovered the lesions of abortion disease in guinea-pigs inoculated with samples of milk from the District of Columbia supply, and in guinea-pigs inoculated with milk from one Experiment Station cow that was not in contact with the healthy herd, but we did not know the cause of the lesions. It was not till sometime afterward before we isolated a bacillus from the lesions in the guinea-pigs, and another interval of time elapsed before the bacillus was positively identified as the etiological factor of infectious abortion in cattle.

The occurrence of the abortion bacillus in the milk of infected cows is now supported, not only by the evidence supplied by Cotton and myself, but also by the work of Theobald Smith and Marshall Fabyean

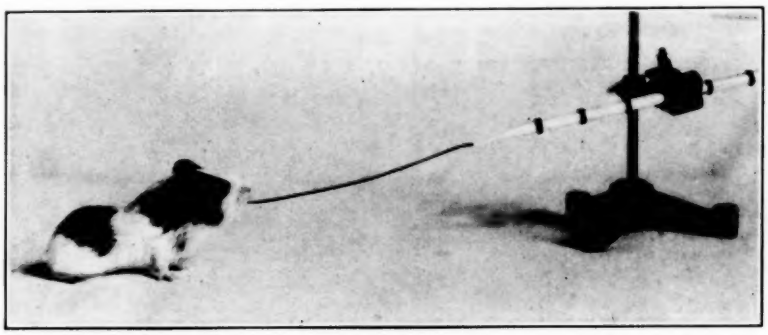

Fig. 3.—A young guinea-pig thoroughly acquainted with its artificial foster mother.

and others. In our work at the Experiment Station we have found ample reasons for believing that many, if not most, cows that become infected with the abortion bacillus remain chronic carriers for long periods of time, probably permanent carriers, and that the bacillus is of common occurrence in the milk of cows that have ceased to abort and have produced several normal, healthy calves in succession. We have records of two cows, one the mother of eight and the other of three calves, the milk of which was infected, though neither had ever aborted. Among aborting cows the bacillus appears in the milk before an abortion occurs, and in one instance was discovered in the milk of a cow that did not abort until 255 days later.

It must be clear from this that the bacillus of infectious abortion of cattle is a factor we cannot afford to omit from consideration when our choice lies between the use of raw and heated milk. We don't know as yet what effect the bacillus has on human health, but we do know, if the

general literature on the subject is reliable, that it is pathogenic for widely different species of animals, and that it is a remarkable organism because of the long time it may linger in the bodies of its hosts with undiminished virulence, and that long periods of time may elapse between its entrance into the bodies of its victims and a manifestation of the disease it causes.

Infectious abortion has become so common among cattle, and seems to be spreading so rapidly, that I have come to the conclusion that its influence on animal industry, from the strictly economic stand, is even more to be feared than that of bovine tuberculosis. In a series of milk tests made at the Experiment Station, in which 516 samples of milk from 90 dairies were injected into 1,068 guinea-pigs, 103 guinea-pigs became affected with abortion disease, and the milk distributed by 29 dairies was proved to be infected, from time to time, with abortion bacilli.

But to return to the breeding record of our guinea-pigs in the milk feeding investigation: When the guinea-pigs were killed at the end of the investigation and examined post mortem, it was found that several of those in the raw milk group showed lesions of abortion disease. Now, as abortion disease in cattle interferes somewhat with conception, it is possible that the smaller number of litters born by the raw milk guinea-pigs may have been due to the same cause, although we are not sure of this. Only one among the fifteen raw milk females used in the breeding tests showed lesions of abortion disease, and the infrequency of the disease among the guinea-pigs of the group generally indicates that the abortion bacillus did not become a factor until the milk feeding part of the tests was near its end. Young born dead were no commoner, but formed a greater proportion of the total number of young produced, among the raw than among the pasteurized milk guinea-pigs. The slightly greater productivity of the boiled than of the pasteurized milk guinea-pigs is important mainly because it gives us still another record in which boiled cow's milk is proved superior to raw and pasteurized milk as a food for young, unweaned guinea-pigs.

The first lesson the investigation teaches is, that the artificial use of the milk of a foreign species, as a food for unweaned animals, is a more lasting handicap than it is generally believed to be. The human organism is infinitely more complex than that of the guinea-pig, and may, therefore, suffer in many ways that cannot be demonstrated by tests with guinea-pigs.

The second lesson is that it is constantly becoming more and more apparent that we lack the means to produce cow's milk that is constantly free from pathogenic agents, just as the number of instances are increasing which prove that seemingly healthy individuals often are dangerous disseminators of disease germs.

The third lesson is that the time has come to ask, when a foreign milk is substituted for the milk natural to the species, whether the modifying effect of heat, irrespective of the treatment living pathogenic contaminations require, may not be beneficial rather than objectionable?

Finally, it is my sincere hope that no one will conclude from my paper that I undervalue the magnificent work of the American Association of Medical Milk Commissions. Milk is a cause of much disease, and there are several kinds of milk that are responsible for this charge. Dirty, stale, adulterated or otherwise objectionable milk, raw, pasteurized or boiled, is and remains a menace to health. Heat, applied to milk, is not a renovating, rejuvenating or a cleansing agent; all we can expect of it is to neutralize those specific germs of disease that find their way into milk, which is the best culture medium for bacteria among all articles of food, in spite of our utmost care to exclude them.

Infected milk? Unless I am greatly in error, I believe those who have studied the question impartially cannot be far from the conclusion that the infants who must, unfortunately, be fed artificially on cow's milk or the milk of a foreign species, and invalids whose diet is restricted as a whole or in part to milk, should receive properly certified milk efficiently pasteurized, or preferably, boiled, and that the general milk-supply should be made as good as the economic conditions affecting the production and handling of milk permit, and should be properly pasteurized, or preferably, boiled, under official supervision.

THE ROENTGEN RAY IN PYLORIC OBSTRUCTION

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Much information has been gathered since the study of the stomach was begun with the Roentgen ray in conjunction with bismuth. In the adult the size, position, shape, function and presence of tumors may be ascertained with a great amount of accuracy. In the baby, however, the conclusions arrived at in the study of the adult stomach are of limited value. Some work has been done, but much more is yet to be done before the information obtainable by means of the Roentgen ray in the study of the baby's stomach will be had.

The Roentgen ray has revealed to us the erroneous impressions we have entertained, more especially regarding the position, size, shape and function of the baby's stomach. Further studies will show, among other things, the influence of the quantity, quality and composition (including the elements and reaction) of the food given on the function of the baby's stomach. The time the stomach begins to empty itself has been shown to be much earlier than was thought, and the presence of a given feeding has shown that it remains in the stomach longer than we believed. On the other hand, the Roentgen ray with the aid of bismuth corroborates our former clinical observations as to the effect of high fats in retarding the emptying of the stomach. Further study with reference to the causes which influence the various reflexes of the stomach and duodenum will be of interest in illustrating the work already done by the physiological chemist. It will be seen, therefore, that much is yet to be done in the study of the normal stomach before definite conclusions can be determined.

By closer attention to the Roentgen ray and bismuth observations in the examination of the infant's stomach, it is possible that a means of differential diagnosis will be obtained which will be of value, especially in young infants, and more particularly in conditions in which obstruction of the pylorus is suspected. It may also explain certain phenomena which make the differential diagnosis between pylorospasm and hypertrophic pyloric stenosis difficult.

While the diagnosis of pyloric obstruction is an easy one to make, it is at times difficult to differentiate between a severe pylorospasm and an hypertrophic pyloric stenosis, and when it is considered that time is a

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* Read in the Section of Diseases of Children of the American Medical Association, at the Sixty-Fourth Annual Session, held at Minneapolis, June, 1913.

vital element in some instances every means to corroborate a diagnosis is essential. It is believed by some that in a marked case of pylorospasm with symptoms of loss of ground, it should be treated surgically, while others do not concur in this belief. However, if we are reasonably certain that pylorospasm is the condition under consideration, procrastination can better be practiced in the adjustment of food than in stenosis. Before arriving at any definite conclusions in the differential diagnosis by means of the Roentgen ray with the aid of bismuth in these two conditions, it will require a study of many typical cases in which absolute proof is shown either at subsequent operation or necropsy that a tumor is or is not present; and in order that these observations may be of the greatest value they should be made in some uniform way. By this I do not mean that all cases should go to operation in order to secure the information desired, but even in those cases in which the infant progresses satisfactorily much information may be obtained. Not only should the observations be made early, but also later in childhood and in after-life, in order that a better insight into the prognosis of these cases may be had.

There is no doubt that there are two distinct conditions which may cause an obstruction, namely, a pylorospasm and an hypertrophic pyloric stenosis, and that either condition may exist alone. Evidence is plentiful to show also that an hypertrophic pyloric stenosis may have associated with it spasm of the pylorus. The obstruction caused by the hypertrophic pyloric stenosis may vary, as Cautley[1] says, "from a degree which is fatal unless relieved surgically to a slight hyperplasia which is compatible with life."

Various theories and views have been advanced for the etiology of pylorospasm and of hypertrophic pyloric stenosis, but as these do not concern this paper a discussion of them will not be considered. Suffice it to say that pylorospasm is a functional obstruction of the pylorus, while hypertrophic pyloric stenosis is an organic obstruction due to an hypertrophy of the circular muscular layer of the pylorus, and at times also a slight thickening of the longitudinal muscular layer, with the mucosa swollen and thrown into longitudinal folds.

When the pylorospasm is severe it produces the effect of an hypertrophic pyloric stenosis, while if the hypertrophic pyloric stenosis is mild, but associated with pylorospasm, it may simulate a simple pylorospasm. The inconstancy in the severity of the symptoms in a case of hypertrophic pyloric stenosis is explained in the latter way. In pylorospasm the symptoms cease when the cause is removed, but in hypertrophic pyloric stenosis the obstruction is permanent. Since the hypertrophic pyloric stenosis is a permanent condition, and since many cases are

1. Cautley: Brit. Med. Jour., Oct. 13, 1906, p. 939.

greatly handicapped for life by the obstruction, and since operation does not interfere with the chemism of the gastro-intestinal tract, surgical attention should be promptly given in those cases needing it.

The greatest difficulty lies, as has been said, in differentiating a severe pylorospasm from a mild hypertrophic pyloric stenosis, the reason being

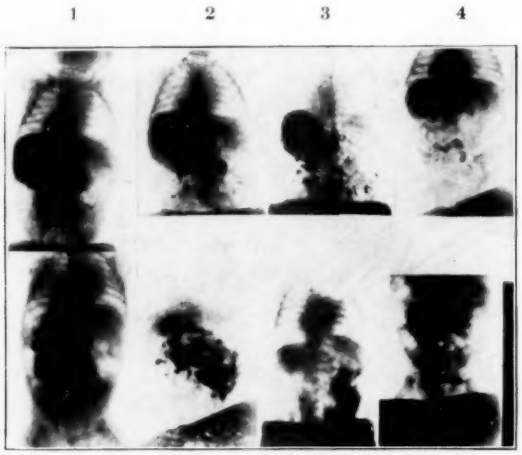

Fig. 1.—Charles. Taken immediately after giving food by gavage. In this and the following illustrations, the plate was in front, and the tube behind the patient, so that the roentgenograms are viewed postero-anteriorly.

Plate 1 (9:47½ a. m.): Note shape of the stomach and the waves. The stomach is squeezing the food out. Duodenal stream is clear, as is also stream passing through pylorus. Some food has already left the stomach.

Plate 2 (9:57½ a. m.): Note waves, also duodenal stream broken.

Plate 3 (10:07½ a. m.): Note waves. Duodenal stream visible.

Plate 4 (10:22 p. m.): Note waves. Colon can be clearly seen filled with gas.

Plate 5 (12:04 p. m.): Baby has been fed. Note that the bismuth meal is being forced out by the new feeding which appears as absence of shadow. Duodenal stream is plainly seen.

Plate 6 (2:50 p. m.): Residue in stomach. Bismuth has reached the ascending colon.

Plate 7 (5:04 p. m.): All bismuth out of small intestines and in ascending transverse and splenic flexure of colon.

Plate 8 (8:55 a. m., 23 hours after): All bismuth has been passed. Colon is outlined with gas.

the similarity of symptoms. The onset, vomiting, peristalsis, constipation and loss of weight, are found in both. A tumor may be palpated in spasm, and it may not be palpated in hypertrophic pyloric stenosis. Cumston[2] states that pyloric tumor was detected in only 25 per cent. of

2. Cumston: Interstate Med. Jour., April, 1911. xviii, 401.

a long list of cases studied, and Cautley[3] states, "I have seen cases in which the pylorus was said to be distinctly palpable and yet no hypertrophy found after death." Morse[4] states that "it is never safe to conclude that there is no tumor, however, unless the abdomen has been examined with the stomach, both full and empty, and with the abdominal walls relaxed, if necessary, under an anesthetic." It would seem, therefore, that some means other than the palpation of the pylorus is needed to aid in the differential diagnosis of the two conditions causing pyloric obstruction. From observations already made the Roentgen ray promises to fill the need which is so urgent. By its use we will be better able to say that this case should, and this case should not, be surgical, and it is very likely that the mortality, which is now about 50 per cent., will be greatly reduced because the babies will be referred to the surgeon before they become bad surgical risks. It is safe to prophesy that the Roentgen ray will become a routine measure, as it should be, in the diagnosis of cases of pyloric obstruction.

The ultimate outcome of cases of pyloric stenosis is variable. They must be considered from two standpoints: those operated on and those not operated on. In the first group are those who survive the operation and escape the complications which arise incident thereto, recover, and continue as healthy normal children, though the pylorus, unless a pyloroplasty is performed, remains the same, as has been shown later by autopsy (Morse-Murphy-Wohlbach[5]), and the food leaves the stomach through the stoma as is seen in the cases of Scudder.[6] I know of no instance in which it has been shown that the food leaves the stomach through the pylorus following gastro-enterostomy for complete obstruction. In other words, if the obstruction is so complete as to necessitate operation it remains so. In the second group are: (a) those borderline cases in which the patients have escaped operation, but who would have been much better off had they been operated on; they remain puny and much below par, have occasional gastric upsets, and are inclined to suffer later from gastric dilatation; (b) those patients who have had marked symptoms due to a spasm engrafted on the stenosis and who, when the spasm has subsided, convalesce and become normal children, the stenosis being only of degree; and (c) those children who may have had slight symptoms, but, under the proper care, have convalesced entirely.

With the modern appliances it is not necessary to anesthetize the child for radiographs, as instantaneous pictures can be made; and when it is necessary, an intensifying screen may be used. The observations

3. Cautley: Brit. Jour. Child. Dis., May, 1908. Reprint.
4. Morse: Am. Jour. Dis. Child., May, 1911, i, 373.
5. Morse-Murphy-Wohlbach: Boston Med. and Surg. Jour., 1908, clviii, 480.
6. Scudder: Surg., Gyn. and Obst., 1910, xxi, 275.

should be made under as nearly normal conditions as possible. The meal should be at the regular feeding hour and consist of, besides the usual food, a quantity of subcarbonate of bismuth equal to 10 per cent. of the quantity of the meal. If the baby is breast-fed the milk should be withdrawn from the breast, the bismuth mixed with it, and, as in the artificially-fed baby, it should be given by gavage. In this way observations can be made immediately after feeding. If the average capacity of the stomach for the different ages is remembered, and the size of the

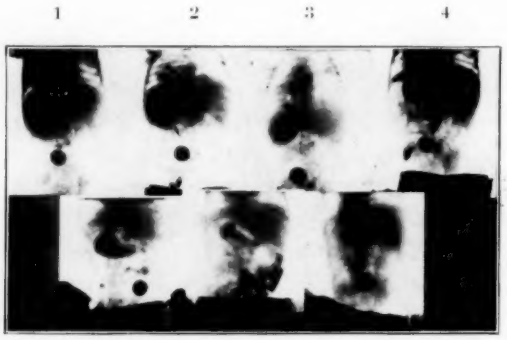

Fig. 2.—Amelia.

Plate 1 (11:16 a. m.): Note waves. Retort shape stomach. Peculiar circle at pylorus. Some food (bismuth) has left the stomach.

Plate 2 (11:26 a. m.): Note waves. Very small quantity of bismuth has left the stomach. Note fine duodenal stream.

Plate 3 (11:35 a. m.): Very little has left the stomach. Note waves are well marked.

Plate 4 (12:15 p. m.): Note waves. Shadow still to left of median line.

Plate 5 (1:03 p. m.): Stomach has become relaxed and assumed transverse position. Baby is turned a little, which may give opportunity for faulty reading of plate as to position.

Plate 6 (3:05 p. m.): Bismuth still in stomach. Gas in stomach marked. Bismuth in cecum and ascending colon.

Plate 7 (9:10 a. m., twenty-two hours later): Bismuth expelled from bowel. Gas in stomach well marked. Outline of colon well seen.

meal made accordingly, the stomach will not be overdistended and the picture will appear as under normal circumstances. The bismuth does not affect the function of either the stomach or the bowels, and I have found that the number of stools per day was neither increased nor decreased.

The position in which the baby is to be placed may be either vertical or horizontal. While the vertical position may give better information because of bringing the viscera covered by the liver more into view,

observations made on young infants in this position are made under abnormal conditions. In the prone position the baby may be placed on his abdomen or on his back. Observations in the former position will also be under abnormal conditions, but if the baby is placed in the latter position as he usually lies after feeding, the observations are more apt to be natural. Of course, it is essential that the detail should be brought out so that if it be necessary the position should be changed to obtain the best results, always endeavoring to have the conditions as nearly normal as possible.

The information to be derived from the radiographs regarding the stomach is the position, shape, size, size of flow through the pylorus when the flow begins, and the length of time required to empty the stomach completely. An absence of shadow is also significant.

The stomach occupies the left hypochondriac region and extends through the epigastric region to the right where the pylorus is overlapped by the liver. Its shape is variable as has been shown by Pisek,[7] and may be of the Scotch bag-pipe, tobacco-pouch, pear, or retort shape. The adult fish-hook type is found only after the second year. The position in which the observations are made may influence the shape, as is shown by comparing the radiographs of Pisek[7] with those of Ladd.[8] The stomach contracts in a particular manner in the normal infant stomach: instead of the peristalsis which is seen in the adult, the stomach contracts in all directions in a squeezing manner. Nature seems to provide against any injury to the stomach by having an air space which acts in a pneumatic way. It is possible that its presence may be the explanation of the projectile vomiting of the baby when there is an obstruction at the pylorus. The size of the stomach varies according to age under normal conditions, and it may be seen dilated or contracted under abnormal conditions.

The rapidity with which the food leaves the stomach is surprising: frequently by the time a meal is finished and an exposure made, some food has already escaped from the stomach. Position of the body does not seem to affect it. It is not always possible to determine the size of the flow through the pylorus, probably because the flow of food is not continuous. The food remains in the duodenum only a short time, passing through spasmodically so that the duodenal stage may also be missed. The length of time required for the stomach to become completely empty has been shown to be variable. Leven and Barret,[9] in a large number of infants from 2 to 6 months old, found the time to

7. Pisek: Trans. Am. Ped. Soc., 1913.

8. Ladd: Trans. Am. Ped. Soc., Washington, May 9, 1913.

9. Leven and Baret: Radioscopie Gastrique et Maladies de l'Estomac. 1909, p. 173.

vary between one and three-quarter hours to two hours for both woman's and cow's milk. Pisek[7] believes the probable emptying time to be less than three hours, and the average final emptying time to be three hours. There are many factors to be considered, however, which influence the emptying time of the stomach. Ladd[10] states that the stomach appears to empty itself of the greater part of its contents from one and one-half to two and one-half hours and that the residue is emptied very slowly,

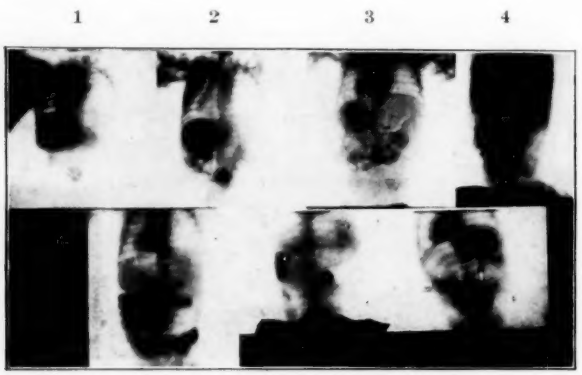

Fig. 3.—Lilian.

Plate 1 (10:10 a. m.): Note stomach in transverse position. Evidence of waves. Nothing has passed into intestines.

Plate 2 (10:15 a. m.): Note hour-glass appearance of stomach. Ball of bismuth being expelled. Gas probably intermingled with bismuth. Bismuth in small intestine.

Plate 3 (10:25 a. m.): Note waves. Hour-glass shape of stomach. Ball of bismuth being expelled.

Plate 4 (10:38 a. m.): Note four waves forcing forward balls of bismuth.

Plate 5 (12:33 p. m.): Bismuth in ascending, transverse and descending colon. Gas in stomach well marked. All bismuth out of stomach.

Plate 6 (3:00 p. m.): Gas marked in ascending and transverse colon. Bismuth in descending colon. Gas in stomach well marked.

Plate 7 (8:50 a. m., 23 hours later): Stomach outlined with gas.

often after remaining for four, five or even seven and one-half hours in both breast-fed and bottle-fed babies. An interesting point is that the residue from the bismuth meal does not appear to mix with a subsequent meal given three hours afterward. Experiments of Ladd[11] show that high fat, fat-free milk and milk with a high protein content retard the emptying of the stomach.

10. Ladd: Am. Jour. Dis. Child., 1913, v, 349.
11. Ladd: Am. Jour. Dis. Child., 1913, v, 357.

In obstruction of the pylorus the radiographs show that the shadows are frequently different from those of the normal stomach in size, shape and position, but more particularly the serial radiographs show the greatest differences to be in the function of the stomach. Observations recorded have been very few, but from the study of them the following may be said: In spasm the shape may not be altered, and in stenosis also the stomach may not be altered in shape, but at times it appears as a round ball (Murphy[12]); both conditions may give the characteristic waves of obstruction; in spasm the position may be the same as in the normal stomach, but in stenosis the shadow of the stomach is frequently limited to the left side and there is no shadow in the pylorus or duodenum; if the stenosis is not complete the stomach will appear as in spasm; the function of the stomach in spasm is changed in a degree, that is, the food may be slower in beginning to leave the stomach and the stomach is emptied spasmodically; it also may require a little longer time for the stomach to empty, whereas in stenosis the food leaves the stomach extremely slowly in some cases and not at all in others, depending on the degree of the stenosis. In Ladd's[13] case a very small quantity of the bismuth was seen in the intestine in two hours, and in Scudder's[14] case no bismuth had left the stomach in four hours.

In only three of four cases coming under my observation during the past ten months have I been able to obtain radiographs. While the number is small I believe that each case is of sufficient interest to report.

CASE REPORTS

CASE 1.—Charles was born on Sept. 9, 1913, sixteen days before he was expected, and was delivered by abdominal cesarean operation. He weighed 7 pounds 10/16 ounce. He was seen by me on the day of his birth. The family history was negative, with the exception of a miscarriage due to a fall. The first day the baby was given water, which he vomited. On the second day he was given 1 ounce of a 6 per cent. sugar of milk solution at two-hour intervals, which he vomited at times. On the third day he was put to the breast. He frequently vomited the breast milk. As the mother had very little milk and the baby was losing weight, a complemental feeding consisting of a 1 per cent. fat, 7 per cent. sugar, .90 per cent. protein and 6 per cent. alkalinity was given. The baby did not seem to care for his food and at times refused to take it. His first yellow stool was passed on the eighth day. His weight at this time was 6 pounds 14 ounces. Not knowing just what he was getting, observations were made to determine how much milk he obtained from his mother. By weighing him before and after nursing it was found that he would get from ¼ to ½ ounce at each feeding. A sufficient quantity of the complemental feeding was given to meet his requirements. This method of feeding was pursued until the sixth month, the quantity and strength of food being increased from time to time, always approximating the capacity of the stomach. The alkalinity was lime water equal to 50 per cent. of the amount of milk and cream. From the time this was

12. Murphy: Surgical Clinics of John B. Murphy, 1913, ii, No. 1.
13. Ladd: AM. JOUR. DIS. CHILD., 1913, v, 355.
14. Scudder: Surg., Gyn. and Obst., 1910, xxi, 285.

begun he commenced gaining and at his sixth month weighed 14 pounds 6 9/10 ounces.

On September 16 typical peristaltic waves were noticed, and while the explosive type of vomiting became more pronounced, no tumor was positively palpated. From this time on the peristaltic waves and explosive vomiting became more and more characteristic. At times he would vomit over the side of the crib to a distance of 3 or 4 feet. On September 21 a crowing sound was first heard when he was nursing. It became significant later as indicating that his stomach was full, as evidenced by explosions if nursing was continued. By careful feeding, particularly as to composition, alkalinity and quantity, it was possible to minimize the number of vomiting attacks, so that he frequently would have only one a day. His weight was noticed carefully and showed a daily gain. His stools were normal, there being only one daily. The explosions continued from time to time, gradually becoming less frequent until January 18, when the last one occurred. The waves became less and less evident as the baby became fatter. A discomfort noticed after feeding when the waves were apparent, however, continued after the waves could no longer be seen.

Jan. 19, 1913, the radiographs herewith presented were made. His usual feeding of 6 ounces of a 1.5 per cent. fat, 7 per cent. carbohydrate, 1.29 per cent. protein was given, to which was added three teaspoonfuls of subcarbonate of bismuth. He was placed in a horizontal position on his back. As will be seen, his stomach is functioning nearly normally. Bismuth was found in his stool five hours after the bismuth was given him, and he had one stool as usual on that day.

At present the baby is apparently healthy and his stomach functions normally. When last seen at the age of 7½ months he was taking 8 ounces of a 3 per cent. fat, 7 per cent. carbohydrate and 2.10 per cent. protein at three-hour intervals, and a cereal jelly once daily. He weighed 16 pounds. On one or two occasions when he had some acute infection of his nasopharynx a mercurial purgation followed by castor oil was given and was well borne.

Case 2.—Amelia was seen in the out-patient department of Touro Infirmary on Dec. 17, 1912, when she was 5 weeks old. She was born at full term and weighed 7 pounds. There was a luetic family history. She was breast-fed, but had frequently vomited since birth. Her weight when first seen was 8 pounds 4¼ ounces. Her stools were black until the third day. She was small, had a wig, the posterior bosses were prominent, and there was scaling of her palms and soles. Otherwise the examination was negative. On December 21 the Wassermann was negative. She had been having three to four green, watery stools daily. She improved slowly, vomited occasionally, and her stools improved in character and decreased in number. She was weighed before and after nursing the breast and showed a gain of only 2½ ounces. At her third month she was constipated. She vomited after, and also between, nursings. March 18 the mother reported that the baby vomited frequently, the vomiting being explosive in character. She was having three to four stools daily, and had lost weight. Her weight was then 9 pounds 8 ounces, having lost ¾ pound since February 24.

Peristaltic waves could be seen, but owing to the rigidity of the abdominal walls palpation was unsatisfactory. No tumor was felt. She got only 2 ounces at each nursing at this time. She was ordered 6 ounces of a weak mixture of a 1 per cent. fat, 7 per cent. carbohydrate, .90 per cent. protein, and 6 per cent. lime water every 2½ hours. March 25 she did not retain the food, had green stools and explosive vomiting continued.

April 5 the Wassermann was positive. The following day the mother was given 6 decigrams of salvarsan and the baby was allowed only breast milk.

April 8 radiographic observations were made. She was given 6 ounces of milk mixture with three teaspoonfuls of bismuth subcarbonate and the accompanying radiographs were taken. She was placed in the horizontal position on

her back. Bowels did not move until twenty hours after the observations were made and bismuth was found in the stool.

April 26 no explosions since mother was given salvarsan, and since inunctions of mercury were begun two weeks before. At present the baby is continuing to improve, takes all food and is much better in every way.

CASE 3.—Lilian was born Feb. 16, 1913, at term. The confinement was normal and the baby weighed 6 pounds 12 ounces. She was first seen March 4 because she was not doing well. She was breast-fed and vomited at times. She had several green, watery stools daily. She weighed 6 pounds 12 ounces. Family history was negative and physical examination showed a small baby with pinched expression, poorly nourished. Otherwise the examination was negative. She was given a complemental feeding of 2 ounces of 1 per cent. fat, 7 per cent. carbohydrate, .90 per cent. protein and 6 per cent. alkalinity.

The infant was again seen March 11 when I was called on account of the illness of her brother. Because of the baby's appearance the mother was instructed to weigh her before and after nursing in order to determine the quantity of food that the baby was getting, and to continue the complemental feeding, because she had gained only 3 ounces since March 4 and apparently was not thriving. For various reasons the observations were delayed and it was not until March 21 that she was seen again. An examination of her mother's milk showed 6 per cent. fat and specific gravity 1.030. The symptoms became more marked and on March 22 a typical explosion was seen. It was said that she had had such explosions before, the first explosion having occurred shortly after her birth. At this time she weighed 7 pounds 12¾ ounces. Peristaltic waves were seen. The breast-milk was ordered withheld for a few days in order to determine whether it was the cause of the vomiting, and the baby was given the complemental food as already ordered, except that 4 ounces were given instead of 2 ounces, with lime-water equal to 50 per cent. of the amount of milk and cream, every two hours. She began gaining, though very slowly, and on March 31 weighed 8 pounds 1 3/8 ounces. The symptoms continued and became more marked, there were frequent explosions, and peristaltic waves were constantly seen during and after feeding. Her bowels moved six times daily; stools well digested but soft. The mother had allowed her breasts to stop secreting milk. A wet-nurse was considered, but because of the radiographic findings and the gain in the baby's weight artificial food was resorted to exclusively. A satisfactory palpation of a tumor was obtained.

On April 6 she was given 3 1/3 ounces of her regular formula (which was at the time a 1.33 per cent. fat, 7 per cent. carbohydrate, 1.20 per cent. protein, 17 per cent. lime-water), with two and one-half teaspoonfuls subcarbonate of bismuth. The radiographs herewith presented were then made. She was placed in a horizontal position on her back. Bismuth was found in the stools three hours after it was given. She had six stools, as usual, the day of the observations.

At present she is progressing very satisfactorily, having gained 1 pound 8⅛ ounces in the previous twenty-two days. The last explosion occurred on June 1, but waves can be seen during and after each bottle. The cinematograph pictures were made June 12, 1913. She is now taking 5 ounces of a 1.80 per cent. fat, 22.5 per cent. carbohydrate and 1.8 per cent. protein at each feeding, and is having two perfect stools daily.

DISCUSSION

Early in the case of Charles (Case 1) I was inclined toward the belief that he had a congenital hypertrophic pyloric stenosis because of the history, the promptness with which the symptoms began, the constipation, the projectile vomiting and the peristaltic waves. The loss of weight during the first three days was not extreme, but I believed that it was due to having begun promptly with the feeding. The inability to

palpate the tumor may have been due to the fact that there had not been much wasting with the consequent thinning of the abdominal walls, making palpation more satisfactory. However, realizing the infrequency of palpating the tumor, failure to find it was not considered unusual. The slow gain in weight may have been found in other conditions. At 1 month the baby weighed 8 pounds, or 8¼ ounces more than at birth, which was believed to be satisfactory considering existing conditions. As the baby improved, the symptoms becoming less marked (though they persisted); the possibility of a pylorospasm being the cause of the symptoms was not lost sight of. On January 19, though the baby was progressing particularly well, it was decided to determine the function of the stomach. The evidence obtained led me to believe that the symptoms may have been due in great part to spasm of the pylorus because of the excellent functioning of the stomach; but the continuation of peristaltic waves and the prolonged stay of the bismuth in the stomach (more than five hours) were sufficient to warrant a diagnosis of hypertrophic pyloric stenosis a degree with probable spasm.

In the case of Amelia (Case 2) the feeding, vomiting and stools would suggest a digestive disturbance, but the projectile vomiting and peristaltic waves, with the other symptoms, would indicate a pylorospasm. However, the luetic condition may have played an important rôle in this case. The slowness with which the food left the stomach, the length of time it remained in the stomach, and the waves suggest an obstruction of the pylorus. The fine duodenal stream may also be indicative of some obstruction. The peculiar circle in the region of the pylorus and duodenum may be significant. The radiographs shown in this case, the symptoms, family history, personal history, and the amelioration of the symptoms coincident with the beginning of antiluetic treatment (the baby taking food after, which before the treatment she could not take), strongly point to the luetic condition as being an influencing factor at least in producing the obstruction (probably pylorospasm) which was the cause of the symptoms in this case.

In Lilian's case (Case 3) the condition is one more especially of pylorospasm though there may be a degree of hypertrophic pyloric stenosis. The onset of the symptoms, the characteristic vomiting and peristaltic waves are indicative of some obstruction. The frequent stools would suggest a spasm, but the presence of a palpable tumor, while it may be rarely felt in spasm, is more suggestive of an hypertrophic pyloric stenosis. However, with the evidence obtained with the Roentgen ray, the position of the stomach, the early outflow of bismuth and emptying of the stomach, the condition is undoubtedly due in a great measure at least to pylorospasm.

I wish to express my thanks to Dr. E. C. Samuel for the radiographs presented in this paper.

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VULVOVAGINITIS IN CHILDREN *

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A striking change has been evident in hospital and medical practice within the last ten years. The advent of social service work in the hospital routine has resulted in a new conception of many disease conditions. Certain diseases have been long recognized as intimately connected with conditions of living, and steps have been taken toward the prevention of such diseases and the protection of the community from the spreading of the infection. Tuberculosis stands as a notable example of this group. Other diseases until recently considered as presenting almost entirely medical problems, are now recognized as having a large social element. As yet sufficient attention has not been given to a study of the social aspect of these diseases. Vulvovaginitis in children belongs in this class. This disease is confined largely to hospital practice, which would at once suggest that it is dependent in part, at least, on poor social conditions. In the Children's Medical Out-Patient Department of the Massachusetts General Hospital we have been studying these cases from the social as well as the medical point of view.

CLASSIFICATION

The ordinary classifications[1] of vulvovaginitis in children recognize a non-specific vaginal discharge associated with certain infectious diseases, or with local inflammatory processes, such as herpes and impetigo. Such cases are not considered here. Another group includes certain vulvovaginal infections in which the etiologic organism appears to be some other bacterium than the gonococcus. A few such cases have been noted in our clinic. The exact bacterial etiologic factor in these cases undoubtedly varies in different individuals. Genito-urinary surgeons recognize certain cases of urethritis in the male which show profuse creamy discharge and are very resistant to treatment, and in which an organism morphologically similar to the gonococcus—the micrococcus catarrhalis — is found. Two of our cases were probably in this group. The third group of cases of vulvovaginitis includes those of true gonorrheal infection. Thirty of these cases have been studied.

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*Read in the Section on Diseases of Children of the American Medical Association, at the Sixty-Fourth Annual Session, held at Minneapolis, June, 1913.

* From the Children's Medical Department, Massachusetts General Hospital.

1. Rotch, Holt, Kerley, Koplik and others: Text-Books on Pediatrics.

DIAGNOSIS

The diagnosis of gonorrheal vulvovaginitis is usually made from the finding of morphologically typical gonococci in a stained smear from the vagina. This is open to a possibility of error in certain cases, in that other organisms may simulate closely in appearance the gonococcus. A more definite diagnosis can be made by the means of cultures. The isolation of the gonococcus in pure cultures establishes the diagnosis beyond question of a doubt. The gonococcus is not an easy organism to cultivate. The complement fixation test[2] is now used extensively as another means of diagnosticating gonorrhea infections. Broadly speaking, the fixation test is like the Wassermann reaction and is specific for the gonococcus. Although there are at present certain atypical reactions not perfectly understood, in general the test furnishes a reliable means of diagnosis and in conjunction with the other tests of smear and culture it is of distinct assistance in doubtful cases. We have confirmed our diagnosis in nine cases by this means.

SOURCE

The problem of determining the source from which these children acquire their infection presents a real difficulty. Hospital and school epidemics have been recognized and studied for many years.[3] Fortunately at the present time such epidemics are less frequent than formerly. In these epidemics we are dealing with a known source of infection and have to discover the method of communication from one patient to another within a given institution. This is quite a different matter from determining the source of infection in individual patients who present themselves for treatment in an out-patient clinic. Much difficulty has been encountered in tracing the etiology of these infections. In a report of 344 cases, Hamilton[4] had 45 per cent. of the cases with undetermined sources of infection; and Spaulding,[5] in 83 cases, could not find the cause in 40 per cent. In thirty cases we have been able to determine a possible source for the infection in all but five cases, or 16.6 per cent. In one of these undetermined cases the patient came to the clinic only three

2. Müller: Wien. klin. Wchnschr., 1906, xix, 894; Watabiki: Jour. Infect. Dis., 1910, vii, 159; Schwartz and McNeil: Am. Jour. Med. Sc., 1911, cxli, 693; O'Neil: Boston Med. and Surg. Jour., 1912, clxvii, 464; Smith, G. G.: Am Jour. Dis. Child., 1913, v, 313; Rockwood: Cleveland Med. Jour., 1913, xii, 1.

3. Abt: Jour. Am. Med. Assn., 1893, xxxi, 1474; Baer: Jour. Infect. Dis., 1904, i, 313; Holt: New York Med. Jour., 1905, lxxxi, 521 and 589; Sheffield: Med. Rec., New York, 1907, lxxi, 767; Hamilton: Jour. Infect. Dis., 1908, v, 133; Butler: Interstate Med. Jour., 1910, xvii, 510; Wile: Jour. Am. Med. Assn., 1910, lv, 1473; New York Med. Jour., 1910, xcii, 501.

4. Hamilton, W.: Jour. Am. Med. Assn., 1910, liv, 1196.

5. Spaulding: Am. Jour. Dis. Child., 1913, v, 248.

times and was not thoroughly investigated. One gave a history of using a dirty railroad toilet just prior to the appearance of the discharge. One is feeble-minded. In the other two patients no reasonable explanation for the infection could be found. Four gave a history of assault. One was infected in a hospital. In sixteen cases other members of the same family or persons living in the same house were known to be infected. Four cases were of children who contracted the disease from girl playmates. Twenty-one cases, or 70 per cent., were from preventable sources.

SOURCES OF INFECTION

Undetermined	5	16.6 per cent.
Hospital infection	1	3.3 per cent.
Playmates	4	13.3 per cent.
Assault	4	13.3 per cent.
Other members of family infected	16	53.3 per cent.
Total	30	
Known to be preventable	21	70 per cent.

The statement of these causes of infection immediately suggests the social problems in connection with vulvovaginitis in children. Among the children who contracted the disease from some other member of the family, we could find no instance in which instructions had been given about the care necessary to be taken with the discharge. In many instances the infected person did not know that the disease was contagious by any other than the usual method of infection. It is quite possible that in some instances instructions had been given and the family did not choose to follow them, or did not choose to admit that instructions had been given. In any event, no care was taken to prevent spreading the disease to other members of the household.

A mother said her other children have had the same trouble and she "thinks it runs in the family." Another said an older sister of our patient contracted the disease from a man, but she allowed the two girls to sleep together. A third said she herself contracted the disease from her husband, but our patient was sleeping in the same bed with them both.

MANNER OF INFECTION

We have not tried to determine the exact manner of infection in these children, but it is probably by direct contact,[6] or by the same means as has been shown to be the method in hospital epidemics, by towels, clothes or other material. Efforts made to close this channel of infection would, I think, show as favorable results here as in connection with the pre-

6. Abt: Jour. Am. Mer. Assn., 1893, xxxi, 1474; Cuthbert: Am. Jour. Obst., 1893, xxvii, 698; Westervelt: New York Med. Jour., 1896, lxiv, 748; Schweitzer: Indianapolis Med. Jour., 1910, xiii, 257.

vention of hospital epidemics. I believe that a considerable number of the infections in our children could have been prevented had proper instructions been given to the persons in the family first infected. I believe that we have a definite responsibility to the community in this connection in making the individual understand the importance of the disease with which he is infected and the necessity of preventing its spreading to other people.

The infection contracted from playmates presents another situation which demands the cooperation of a social worker. Bad habits are taught young children by those older than themselves, in some instances by those old enough to understand fully the significance of sexual matters. These habits spread by imitation from child to child. Masturbation is extremely common among the infected cases. Whether as a result of this or not, I am unable to say, but these children are many of them sexually precocious. Just how far this has gone in some instances it is impossible to know, since their precociousness makes them secretive. The usual lack of embarrassment during examination seen in a child of six has entirely disappeared in many of these children. How far we can go in remedying these conditions must remain for further study, but I am confident that as we learn more intimately the history of infected children, the less often we shall have to attribute the infection to toilets and other indefinite sources. The supervision given many children by their mothers is so inadequate that there are long periods of time when they are unprotected from the influences of evil companions. We are driven back, in both the family infections and the infections from child to child, to the necessity of educating parents in their responsibility toward their children.

Prepola was a little girl of 9 years when she first came to our clinic complaining of vaginal discharge. This discharge was proved to be a gonococcus infection. Several days later the mother brought along a small brother aged 7 years with urethral discharge, also gonorrhea. The boy's discharge had been present for some time and antedated the appearance of symptoms in the girl. We concluded, erroneously as it later proved, that the boy was probably the source of infection in the girl. After nearly two years of acquaintance with the family the girl made a confession of the whole matter to our worker. She contracted the disease by direct contact from an older girl who also taught her to masturbate. She in turn infected her smaller brother.

INFECTION IN FEEBLE-MINDED

How important a rôle feeble-mindedness plays in this connection I am not prepared to say, but I am sure it must be considered as a part of the problem. Feeble-minded children are very irresponsible, and apparently particularly so in sexual matters. They are often infected with gonorrhea and are ready to pass on the infection to other children. It is extremely difficult to deal with these children in their homes, and

the fact that they are a source of direct physical injury to other children makes it even more imperative that they should be removed to institutions especially adapted to their care. In five of our patients the infection is directly traceable to feeble-minded individuals, and two other patients were themselves feeble-minded.

A feeble-minded colored child under treatment elsewhere for vaginitis was the inseparable playmate of one of our patients, whose sister also was found to have a vaginal discharge. A mother, who is very low grade mentally, has one child in the state reformatory and two others coming to our clinic with a gonorrheal vaginitis.

The following family history reveals a situation as pathetic as it is serious:

> The younger son of a wealthy family in Russia was sent by his parents to this country because he was "crazy." He was unable to earn a living in his own country. After he arrived here he married and became the father of two children. The older of these children, aged 9 years, is also feeble-minded, but not sufficiently so to prevent her being at large in the community. The father insists on sleeping with this child and this child is found to be infected with gonorrhea. She is perfectly familiar with sexual matters and talks freely of the practices of her circle of playmates, who are boys and girls of her own age. Her most intimate friend is a second patient of ours. The habits of the two girls are such as to make the passage of infection from one to another extremely easy. A third member of the group also has a gonorrheal vaginitis.

In this train of infections—and undoubtedly others in this neighborhood will be found to be infected—the original source of infection should have been removed from the possibility of contaminating others a long time ago. The father's moral influence as exercised through his feeble-minded daughter is quite as injurious as the physical disease which he spreads.

TREATMENT

Published reports[5] of the treatment of cases of vulvovaginitis in children have a very loud note of pessimism. I do not believe that this is justifiable from the facts. We have been able in our clinic through the cooperation of Dr. Hugh Cabot to have an almost ideal arrangement in the management of these cases. They are all retained as patients of the Children's Medical Department, and the home visiting done by our social workers. The actual mechanical treatment of the disease condition in the individual patients is done by a member of the genito-urinary department who is infinitely more experienced in the treatment of these cases than any man practicing general pediatrics.

The treatment employed varied with individual cases, but in general consisted of a cleansing douche followed by the prolonged application of silver salts. There is some difference of opinion as to what constitutes a cure. Certainly the failure to find gonococci in a single smear, or the

temporary absence of a vaginal discharge, is hardly sufficient evidence on which to base a favorable report. It seems to me unfair, however, to refuse to call a patient cured when all symptoms have disappeared and the local examination, including a smear from the depths of the vagina has been negative for a considerable period of time. If these conditions exist and the complement fixation test is also negative, I do not see how there can be any question of the cure. I do not agree with those who believe that the reappearance of the discharge necessarily means a relapse. When the original source of infection still exists and there has been no reform in the habits of the patient a reinfection may easily occur and seems to me a more likely explanation than that a relapse has taken place.

The result of treatment in our thirty patients is as follows: One died in an accident before the treatment was completed. In thirteen the outcome of treatment is undetermined because of the failure to continue attendance at the clinic, due to removal from the city or some other cause. Five of these, however, were reported cured, and have been free from symptoms for a considerable length of time, although no examination of the local condition was made in the clinic. Eight cases are still under treatment varying in duration to date from one month to two years. It is interesting to note that the patient under treatment two years contracted her infection in a hospital. Eight cases are cured. The cure has been proved by a very long period of freedom from symptoms and local irritation or discharge, and in some instances by a negative complement fixation test in addition. A detailed table of these cured cases is given below:

Case	*Under Treatment,* Months	*Without Symptoms,* Months	*Blood*
3	10	3	Negative (culture negative)
4	7	4	No record
5	7	3	Neg.; previously positive
8	21	3	Negative
11	6	6	Neg.; previously positive
18	*	24	No record
27	5	18	Neg.; previously positive
28	1½	17	No record

**By family physician.*

The problem of vulvovaginitis in children will demand careful study for many years, but I believe if we are to make even a beginning toward the eradication of the disease we must recognize that we are dealing not only with a medical disease, but also with a social evil, and that the medical treatment alone is only a part of what needs to be done. This means that we must establish a close cooperation with the social workers in their attempt to solve the delicate questions of venereal disease. It has been our custom in the Children's Clinic at the Massachusetts

VAGINITIS SCHEDULE

Name ..... Age ..... S. S. D. No.....

Address ..... Nationality ..... O. P. D. No.....

Date first visit to O.P.D..... Date diagnosis .... Date referred to S. S. D.....

I. INFORMATION OBTAINED FROM PHYSICIAN.

1. Has smear been made..... Result .....
2. Has fixation test been made..... Result .....
3. Treatment:
   a. How often is patient to come for treatment.....
   b. Any treatment at home.....
   c. Kind of treatment.....
4. Local condition ..... Discharge ..... Irritation.....
5. Is patient allowed to attend school.....

II. INTERVIEW WITH MOTHER OR ATTENDANT AT TIME REFERRED TO S. S. D.

1. Mother's interpretation of situation before explanation.....
2. Mother's explanation of source of infection.....
3. Social worker's explanation of nature of disease to mother.....
   a. How does mother react.....
4. Home:
   a. Other members in family..... Infected .....
   b. Any boarders or lodgers..... Infected .....
   c. Toilet arrangements .....Other families using it.....
   d. Bathing arrangements .....
   e. Sleeping arrangements .....
5. Recreation:
   a. How long away from mother's care during day.....
   b. Where does patient play.....
   c. Form of play .....
   d. Playmates ..... Boys .....Girls .....
   e. Reading .....
6. Habits:
   a. Cleanliness ..... What form .....
   b. Hours of sleeping.....
   c. Hours of eating .....
   d. Typical menu ..... Candy .....
   e. Does patient masturbate.....
7. Nature of patient:
   a. Nervous ..... b. Irritable ..... c. Obedient .....
   d. Confiding .... e. Diffident ..... f. How much has she learned of sexual matters .....
   g. What are her attachments.....
8. Medical social worker's instructions about hygiene:
   a. Precautions against infection of others.....
   b. Food, bathing, etc., of patient.....
   c. Bringing in of others in family for examination.....
9. Name of school teacher..... School nurse..... Physician.....

III. HOME VISIT.

1. Nature of neighborhood and tenement.....
2. Personal inspection of sleeping and toilet arrangements..
3. How is mother following instructions.....
4. Is confidence of mother and patient gained.....

IV. VISIT TO TEACHER.

1. Patient's school record .....
2. Playmates, disposition, etc., as noted by teacher.....

V. SOURCES OF INFECTION.

1. Positive ..... Probable..... Possible.....

VI. RESULTS OF TREATMENT.

General Hospital to refer all cases of vaginitis to a social worker. We have been particularly fortunate in our social worker. She has been able to gain the confidence of the patients and of their families, and as a result it has been possible to find the probable source of infection in a very large percentage of cases. The social records of the vaginitis cases are kept on blanks on which the important medical and social data is collected. As has been done for many years with cases of tuberculosis, we try to secure an examination of all the other members of the family, at least of all the female children. We instruct the mother or other responsible member of the family in the dangers of infection and we visit in the home to see that our suggestions are carried out. We try to make the parents appreciate their responsibility. In almost every instance, when the first infected member of the family was told the true nature of the condition and made to understand the seriousness of the infection, it was possible to obtain full cooperation to prevent further spreading of the disease. The instances in which this was not the case were among families evidently on a low intellectual plane, or among families generally irresponsible for the welfare of their children. One mother, who had been keeping an immoral house, when she understood the situation, moved to a new and clean house and is faithfully doing everything for the benefit of her child. In one instance it was necessary to secure the help of a local board of health to improve the living conditions, but the conditions were improved.

CONCLUSION

In conclusion, may I repeat, that it is possible to find the source of infection in the majority of cases of vaginitis in children. The outlook for cure of the individual patient is good, provided the treatment is followed. To prevent the spreading of the disease, parents must be made to realize the contagiousness of the condition and the dangers in the neighborhood to which their children are subjected, provided they are not protected during the long hours of play. The frequency with which feeble-minded children act as centers of infection gives an additional argument for their isolation. The treatment of the individual patient as she presents herself for our care remains our first duty, but if we do nothing more we can hardly expect to make much of an impression on the disease as it exists in the community.

329 Beacon Street.

PROGRESS IN PEDIATRICS

RÉSUMÉ OF THE LITERATURE ON THE EAR

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The most notable contribution to aural literature during the past year is that of Kopetzky and Haynes on the diagnosis and treatment of otitic meningitis, and this is especially noteworthy in its relation to pediatrics from the fact that a majority of cases thus afflicted are in infancy or early childhood. If the results of Kopetzky's laboratory work are sustained by further clinical observation (of which I have no doubt), they will, in the near future, be adopted as a routine procedure for the early diagnosis of otitic meningitis. Since our chief difficulty in the past has been to make an early diagnosis, before the disease was well advanced, Kopetzky's methods of determining the presence of meningitis should mark a definite advance in the successful treatment of this most fatal of all diseases.

Kopetzky[1] starts his study by a rereading of many published cases of meningitis, covering the entire field of meningeal infection, studying in detail the case histories and autopsy reports, and finds that what is usually differentiated as separate and distinct forms of meningitides, each a definite entity, represent in reality a number of conditions which are fundamentally similar and to a great extent progressive in type. The so-called types vary somewhat regularly in definite relationship to the causal bacterial agent, but the result of the infection of the central nervous system by the microbic invasion produces tissue reactions which are fundamentally similar in all the types.

The author concedes the difficulty in dealing with the question at issue when presenting the argument that meningitis is to a certain extent a progressive disease, in attempting to bridge the gap between the so-called "meningitis serosa" and those meningitides which exhibit frankly purulent characteristics. He calls attention to the rather common experience which clinicians have, of observations made, of clinical pictures which logically are referable only to involvement of the meninges, and in which complete recovery takes place without other therapeutic aid except removal of foci of disease in the tympanic cavity and mastoid process. The performance of lumbar puncture in such cases generally

1. Kopetzky, S. J.: Meningitis. Nature, Causes, Diagnosis, and Principles of Surgical Relief, Laryngoscope, 1912, xxii, No. 6.

presents a fluid found sterile on incubation, and with negative findings as to its cytology.

Kopetzky contrasts the clinical picture and the findings in the spinal fluid in this group of cases, with the clinical picture and laboratory findings from cases of meningitides which so often follow an infectious invasion of the labyrinthine channels of the internal ear — the so-called fulminating type of otitic meningitis — and he comments on the similarity of the findings in the two classes of cases. He reasons that in the so-called fulminating type of meningitis, the lethal outcome is established before frankly purulent characteristics are evident. The fatal outcome is due to paralysis of the vasomotor, the respiratory and the vagus centers in the medulla. On the other hand, in the usual course of the so-called "meningitis serosa," the disease is either aborted through operative measures on the primary foci of disease in the adnexa of the ear, or the case eventually develops the well-known laboratory findings of purulent meningitis. In line with the more recent observations of Körner, the author holds that the serous type of meningitis is, therefore, but the initial stage of what may become a purulent meningitis, and he points out that all of the purulent meningitides pass through this stage, although in the latter instance they are unrecognized clinically. The fulminating type of meningeal infection presents the initial stage, with such a predominance of symptoms from the vagus, the vasomotor and the respiratory centers that the symptoms from the vital centers overcloud the whole picture, and the factors to which he refers are overlooked. Tracing this hypothesis further, case histories and autopsy reports are presented, taken from reliable observers, from published literature, all of which tend to show that the reports of cases descriptive of so-called distinct types of meningitides are, in reality, reports of one and the same tissue reaction studied during a different stage of its development.

Having established the progressive nature of meningeal infections, the author devotes himself almost exclusively to common factors. These common factors are the symptoms of the disease, and the analysis of the symptom-complex of meningitis — keeping in mind only the elements which produce symptoms — resolves itself into a study of the tissue reactions which result from the invasion of the central nervous system by pathogenic micro-organisms. This study of the tissue reactions, besides throwing considerable light on the causes underlying the chemical and other changes found in the cerebrospinal fluid obtained by lumbar puncture from cases of meningitides, presents valuable data, useful in establishing an earlier diagnosis of meningitis than is possible from the means heretofore at our command.

The author first studied the factors presented by brain compression, comparing these with the results obtained by artificially infecting the

meninges. Compression of the brain by increasing the endocranial tension of the cerebrospinal fluid, whether this tension results from hydrostatic means or by bacterial invasion, is the same. It results in a compressed blood-supply. The experiments of Araki, the analyses of Hoppe-Seyler, the observations of Zillessu, and the work of Martin H. Fischer, all point out that the lack of oxygenation of a part, a muscle or an organ results in acid formation. Kopetzky is satisfied that under these conditions, in cases of meningitides, where pressure factors dominate the clinical picture, the cerebrospinal fluid will give evidence of this lack of oxygenation by evidencing a condition of acidity, and the tissues a condition of acidosis.

The observations of Andrew Connell, who remarked on the presence of a "lessened alkalinity" in similar conditions, are of moment. The author advises that the determination of the reaction of the fluid must be made immediately after the withdrawal from the body, for the autolytic changes which the fluid undergoes tend to make it alkaline. This characteristic of the spinal fluid is not specific to fluids from meningitides, however, for the author gives a table showing analyses of fluids obtained from patients suffering from other diseases which produce disturbed intracranial tension, and these likewise present a degree of acidity. The acidity is of the nature termed in chemistry "combined acidity."

The lack of oxygenation is held partly responsible for the finding of lactic acid in the cerebrospinal fluid in meningitides and other diseases which produce a compressed blood-supply in the cranial cavity. The Uffelman reagent is employed in testing for this acid.

The author then proceeds with the study of bacterial activity in the tissues of the central nervous system. He alludes to the fundamental work of Theobold Smith, Arthur Kendall, Lyons and Cramer, Dryfus, Nashimura and many others who have demonstrated that the activity of the bacteria is directly influenced by the presence in the media of their growth of available carbohydrate. The well-established finding that toxins are not produced in the presence of carbohydrate is restated, and then Kopetzky draws attention to the fact that the normal cerebrospinal fluid contains a carbohydrate now generally accepted as a form of non-fermentable dextrose. This constituent of the normal cerebrospinal fluid is the first element to be attracted by the bacterial invasion of the central nervous system, the microbic flora which constitutes the usual infective agent being of that type of micro-organism which prefers carbohydrate to protein in its dietary. Therefore, the first evidence of pathogenic microbic invasion is offered by an absence of this copper-reducing body from the cerebrospinal fluid. There are a few negligible instances where this general statement does not hold true, the most important of which

is the tuberculous infection of slow development. In all other types of meningeal infection, Kopetzky has noted the absence of the copper-reducing body from the spinal fluid obtained from cases of meningitides.

This finding is shown to precede the demonstration of bacteria in the fluid by hours and sometimes even days, and is held to be of great diagnostic value. It absolutely differentiates, according to the author, between any actual infection of the meninges, excepting the slowly-developing tuberculous type, and cases of meningism, meningeal symptoms from sepsis, and other general diseases which present meningeal symptoms during their onset or course. Furthermore, due to factors which the author details, the reappearance of the copper-reducing body later in the course of the disease has always been accompanied by the recovery of the patient, and hence he points out the value of this observation as an aid toward making a prognosis in the case. For the purpose of testing the fluid for the presence or absence of the copper-reducing body, the author recommends the employment of the Benedict solution.[2]

The chief and largest part of the nervous system of man consists in the so-called myelin substances, among which lecithin is the best-known prototype. The decomposition products of lecithin are then studied in the laboratory and compared with the products of this element found in the spinal fluid. Cholin, a poisonous base, is demonstrable in the fluid in larger quantities than is found in what is termed the normal minimum amount. Here the findings of Kopetzky coincide with those published by Gumprecht, Gulewitch, Halliburton and others. Finally, as an end-product, the author found a neutral fat in small percentages. In cases which terminated fatally, this always reached 1 per cent. plus.

Kopetzky concludes this section of his work by emphasizing the necessity of a complete examination of the spinal fluid to get the necessary data for exact diagnosis, and he calls attention to the determination of the so-called "meningeal index" by a method devised by Mayerhofer[3] of Berlin as having been found to be of distinct value.

The examination of the fluid should entail a determination of the pressure under which it is obtained, its physical, cytological and chemical constituents, and finally, its bacterial contents. Kopetzky insists that the chemical examination gives the quickest answer and permits a diagnosis of meningitis before the microbic flora of the spinal fluid is demonstrable.

There are other factors studied; for instance, the influence of the resultant stasis in the circulation of the cerebrospinal fluid, and its effect in permitting the accumulation of the products of cerebral metabolism,

2. Benedict, S. R.: Jour. Am. Med. Assn., 1911, lvii, 1193.
3. Mayerhofer: Berl. klin. Wchnschr., May 5, 1913.

to add to the poisoning of the nerve centers in the cortex; also, the changed consistency in the tissues of the Pacchionian bodies, which hinder the free flow of the fluid to the venous blood-system. These, and the experiments on pure colloid (fibrin) using the normal and abnormal cerebrospinal fluid as media to test out the production of edema, as well as a detailed study of the specific gravity of the fluids in health and disease, form interesting by-problems on which light is shed.

The author summarizes, dividing the symptoms of meningitis into two divisions:

1. Those dependent on increased intracranial pressure.

2. Those dependent on the growth of bacteria, and on the decomposition products thrown into the circulation from the disintegration of nerve tissues.

Kopetzky insists that heretofore we have limited our therapeutic endeavors to combat the disease too much to efforts directed against the bacterial infection and have not made sufficient effort to lessen the intracranial tension, on the one hand, and to neutralize the chemical products of abnormal tissue functionation on the other. He contends that once the pressure factors are in hand, then the added forces which may be brought to bear on the infection itself, in the form of antitoxins, vaccines and chemical agents, will have increased value in the treatment of the symptoms referable to his second division of the symptomatology.

DRAINAGE IN MENINGITIS

For the control of the pressure factors, which manifest themselves clinically by an increasing blood-pressure, by the presence of swelling of an optic disk — not choked disk — and tension under which the cerebrospinal fluid is obtained, Dr. Irving S. Haynes[4] has devised an operation for drainage through the great subarachnoid space known as the cisterna magna, which lies close beneath the occipital bone, accessible to surgical procedure.

When under pressure, the brain is crowded against the skull, and any drain introduced between these two structures is pressed on by the former and quickly walled off by plastic lymph, and so becomes useless. The single exception to this intracranial condition and inevitable result is found at the space between the two poles of the cerebellum and between them and the medulla. This space is the cerebello-medullary angle or hiatus. It forms a large part of the great subarachnoid space known as the cisterna magna. This space is in free communication with all the other subarachnoid spaces about the brain and cord, and also, through a

4. Haynes, I. S.: The Surgical Treatment of Meningitis — Its Scope and Accomplishment, Laryngoscope, 1912, xxii, No. 6; Trans. Ninth Otol. Congress, 1912, p. 355.

funnel-shaped opening known as the foramen of Magendie, with the ventricles of the brain. The cerebrospinal fluid is secreted by the chorioid plexuses of the ventricles and flows from them into the great cistern before taking its final course over the convexity of the brain to enter the sinuses. It is in this region that the infected fluid first gathers, and here the effects of inflammatory activity are most marked. Haynes has shown that the skull may be opened here, even in the presence of great intracranial pressure, without the occurrence of a hernia cerebelli, and that drainage here is free and continuous, because the drainage wick leads from a natural cavity and there are no surfaces to crowd together about it, as exists everywhere else over the brain surface; furthermore, the drain will not be isolated by adhesions, because it lies in a space through which fluid is continually flowing by way of the drain to the surface.

Dr. Haynes summarizes the purposes of his operation as follows:

1. To open the cisterna magna, relieve intracranial pressure and restore the normal supply of blood to the vital centers.

2. To accomplish this without danger of cerebellar hernia or of "corking up the foramen magnum by downward displacement of the brain stem."

3. To prevent shock and possible death of the patient from too sudden escape of the cerebrospinal fluid.

4. To provide for free and continuous drainage of the infected cerebrospinal fluid and thus enable Nature to effect a cure of the disease.

5. To afford inspection of the foramen of Magendie and, if it is closed, allow of its reopening.

6. To forestall possible complications, especially hydrocephalus. Should hydrocephalus be present, its relief and cure may be possible by this operation.

7. To accomplish these desirable ends by an operation of the simplest technic, in the minimum of time, and with the least shock.

Both Dr. Haynes and Dr. Kopetzky insist that the result of the operation will depend on its *immediately* following the *early* diagnosis made possible by the latter's laboratory researches.

TREATMENT

The treatment of meningitis by means of an injection of hexamethylenamin into the spinal column is reported to have given excellent results in some hands, while in others it has absolutely failed and is consequently condemned. For certain reasons the procedure has always seemed to be questionable. As to its efficiency, my own experience makes me very pessimistic. In any event, the result must depend solely on the elimina-

tion of free formaldehyd, and this can only be attained under certain conditions and methods of administration, as shown by the laboratory observations of Kopetzky. I am indebted to Dr. Kopetzky for furnishing me with the results of his recent original observations on the use of hexamethylenamin [urotropin] in the treatment of meningitis. He says:

Urotropin itself possesses but small bactericidal properties. Its effects are only produced when it breaks up into free formaldehyd, and this happens most easily when the chemical reaction takes place in an acid medium. My own work has demonstrated, at least to my satisfaction, that in a certain number of cases acidosis is present in the central nervous system, and a low degree of acidity is presented in the spinal fluids of these patients. The meningitides in which this finding usually occurs are those in which evidences of an increased intracranial tension are most marked. This will probably explain the differing results obtained by one and the same man in a series of meningitides, for all do not exhibit pressure factors.

Urotropin given by way of the stomach is found as urotropin and not as formaldehyd in the blood. It is rapidly excreted in the urine both as urotropin and as free formaldehyd. It breaks up into formaldehyd and other products when passing through secretion-producing cells (epithelial cells of the type found in glands which functionate to produce secretions). The uselessness of directly injecting urotropin into the spinal canal is, therefore, clear. In introducing any product into the spinal canal, many factors must be considered. The fluid normally is a watery, clear liquid with a specific gravity of from 1.005 to 1.007, varying individually. Its salt content is very low and is of the natrium variety. Protein elements are also very low. It would seem as if the physiologic function of passing easily through the Pacchionian tissue and seeping into the venous blood channels necessitated this composition of the fluid. To add a dose of urotropin, 40 to 50 grains, even diluted with sterile water or physiologic salt solution, as I have seen done, is certainly not tending toward the establishment of normal relationship of this fluid to that of the blood plasma with which it is its destiny to mix. In many cases the clinicians employing such a procedure have simply added another factor — an irritant factor, overlooked because of the patient's condition — to the stasis in the circulation of the cerebrospinal fluid which is already present because of the edema of the meninges, including the Pacchionian bodies.

The only secreting epithelial cellular elements within the cranial vault, as far as present-day knowledge of brain metabolism goes, are the cells of the chorioid plexus, and as is seen from the evidence presented from the injection of salvarsan in the treatment of brain syphilis, these cells secrete so little of the salvarsan that a distinct disproportion is demonstrable between the amount found in the blood-stream and the quantity found in the cerebrospinal fluid two hours after the salvarsan injection. (See Rockefeller Institute reports.) These cells of the chorioid plexus, whose function it is to secrete the spinal fluid, must possess an inhibitory action on the secretion of solid matter, whether of basic salt nature or loosely combined protein elements. This must be so to preserve the spinal fluid in its normal density and composition in the face of drugs and diet whose elements are often found in the blood and not in the spinal fluid.

Finally, to force the secretion of the decomposition products of urotropin brought to the cells of the chorioid plexus by the blood, this vehicle must be surcharged with the drug, and in the face of its rapid elimination by the kidneys, this presupposes the employment of a very large dose by way of the stomach.

From experimental work not yet published, I feel that urotropin (hexamethylenamin) offers us the best means of combating bacterial activity when given in large doses, by way of the stomach, combined with cream of tartar. When pressure symptoms are presenting, the local acidosis helps the formation of the formaldehyd. Where pressure symptoms are not the predominating

factor, the large dosage causes increased secretion of the cells of the chorioid plexus.

DEAF-MUTISM

The ever-important question of deaf-mutism, both congenital and acquired, continues to be the subject of many communications. Notwithstanding the improved methods of teaching this unfortunate class, our best efforts must be directed to the employment of prophylaxis. In an interesting communication by MacLeod Yearsley[5] on the prevention of deaf-mutism, he makes the plea that all otologists should turn their attention to the study of deaf-mutism, and feels that the movement for its prevention should be international. The general practitioner generally sees the potential deaf-mute first, and on him rests the responsibility of prompt treatment and advice. Every deaf-mute school contains pupils who are the victims of lost opportunity.

Yearsley has analyzed 1,076 cases, of which 45 per cent. were born deaf and 55 per cent. acquired deafness. Of those born deaf, three groups could be arranged: (1) Those in whom no family history was obtainable; (2) those in whom family history contained no evidence of other deaf births; (3) those in whom there was a family history of congenital deafness. These really may be taken to show that there are cases of true hereditary deafness or sporadic congenital deafness. Some of the second class may have acquired deafness in very early infancy, as deafness is often not detected until the second year. Of the congenitally deaf, 145 cases in 123 families, or 24 per cent., showed deafness in direct line of parentage; 31 per cent. showed it in collateral lines; the collateral family lines are equally as, if not more important than, the direct. Out of 309 families in which there were cases of congenital deafness, cousins married in 7 per cent., and in 592 families in which there were cases of acquired deafness, cousins married in but three tenths of 1 per cent. In one school in which there was a large proportion of Jewish children, out of 137 families, consanguinity occurred in 27 per cent.; thus, combining the two percentages in the 446 families in which deaf-mutism occurred, there were 59 consanguineous marriages.

Alcohol and insanity play an important part in congenital deafness. Syphilis affected more the sporadic cases, according to Yearsley's opinion. Of the 592 acquired cases, 34 per cent. were due to the infectious diseases, 26 per cent. of these belonging to the exanthemata in the following order: Measles, 11.5 per cent.; scarlatina, 9.7 per cent.; diphtheria, 2 per cent.; influenza, 0.67 per cent.; pertussis, 1.18 per cent.; German measles, 0.33 per cent.; enteric fever, 0.33 per cent.; cerebrospinal meningitis, 0.16 per cent.; as also varicella and mumps. Of other diseases, pneumonia

5. Yearsley, MacL.: Ann. Otol., Rhinol. and Laryngol., September, 1912, p. 585.

was responsible for 2.7 per cent.; tuberculosis, 0.33 per cent.; syphilis, 6.5 per cent.; diseases of the nervous system, 18.7 per cent.; and middle ear suppuration and catarrh, 20.7 per cent. Of the cases due to injury, fifty-eight of the acquired cases fell in this division.

Yearsley looks forward to laws prohibiting the marriage of deaf-mutes, blood relations, alcoholics and syphilitics; better care of children generally; more careful examination of the ears in all fevers; the recognition of the evil effects of adenoids and nasal diseases, and better teaching of otology in the medical schools.

The above résumé forcibly illustrates that, after all, preventive medicine in its relation to otology is many times the most effective, and, indeed, the only means of successful treatment. A great majority of all aural diseases and their complications start as more or less simple tympanic affections, and, if promptly treated, will frequently be arrested before any considerable damage has occurred. In other words, the correction of any abnormality in the nasopharynx, such as the removal of diseased tonsils and obstructive adenoid vegetations, and the establishment of a free nasal respiration, will prevent the extension of many pathologic processes from the same through the Eustachian tube into the middle ear. Then, again, as I have pointed out on numerous occasions, if a suppurative infection of the middle ear actually occurs, this should be promptly recognized and the fluid evacuated by an immediate incision of the membrana tympani. By the employment of these simple measures in the hands of the general practitioner, and more especially the pediatrist, most of the serious complications, such as meningitis, sinus thrombosis, brain abscess formations, and even deaf-mutism, will have been prevented.

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